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Oedema and Its Management*

By EDWARD A. BARTRAM, M.D.,
London, Ontario

THE problem of oedema is one that has aroused the interest of physiologists and clinicians for centuries. Although its mechanism is complex and many controversial points remain, recent investigation has shed much light on the subject, so that we are now able to formulate a rational basis for most types of oedema as they appear for clinical study.

Oedema is a physical sign denoting excess fluid in the tissues of the body, chiefly in the intercellular spaces. It may be local or general in extent and when universally distributed loses its identity as oedema and becomes known as anasarca. Under normal conditions the outflow of fluid from the vascular system into the tissue spaces and the inflow of fluid from the tissues into the vascular system and lymphatics is nicely adjusted. Oedema occurs when the balance is disturbed resulting in a greater passage of fluid into the tissue spaces than out of them. It is my intention to discuss generalized oedema and to mention the chief factors concerned in its mechanism.

In order to understand the various types of oedema, we must be familiar with the forces which regulate the passage of fluid back and forth through the capillary wall. The influence of certain of these forces is now clearly understood, that of others is still veiled in mystery.

In the mechanism of oedema, five important factors are involved. The first of these is the filtration or hydrostatic pressure within the capillary which tends to drive fluid into the tissue spaces. This is of primary importance in circulatory failure oedema, either cardiac or hepatic. The second factor concerns the colloid osmotic pressure exerted by the plasma proteins which tends to draw fluid from the tissues into the blood. Lowering of the colloid osmotic pressure is chiefly responsible for the widespread oedema of the "nephrotic syndrome" and the lesser grade of oedema associated with malnutrition. The third factor involves the permeability of the capillary wall, injury of the vessel allowing fluid

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to escape into the surrounding tissues. Inflammatory and anaphylactic oedema arise in this way and it is probable that oedema in the early stages of acute nephritis is the result of a diffuse capillaritis. The fourth factor is the qualitative and quantitative relationship between the electrolytes in the blood and tissue spaces. In certain forms of oedema, changes in the electrolyte pattern exert a profound influence. Fifth, the part played by the lymphatics which drain a portion of the intercellular fluid into the blood stream has received little attention. In addition, a nervous control exists which probably plays its role by exerting an influence over certain of the factors already mentioned. Finally, the relation of the endocrine glands to the mechanism of oedema must be considered, for in certain types they play an important part.

We owe to Starling our knowledge of the importance of colloid osmotic pressure. It was he who first suggested that the exchange of fluids between the tissue spaces and the circulating blood is determined by the relative magnitude of two forces: (1) The hydrostatic pressure which tends to drive fluid from the blood vessels into the tissues; (2) The colloid osmotic pressure exerted by the plasma proteins which tends to draw fluid from the tissue spaces into the blood stream. Starling's theory receives ardent support from many investigators who have devoted considerable time and thought to this particular phase of the problem. It was given practical value by Epstein in explanation of the oedema of nephrosis, in which he found the plasma protein values to be low. It is interesting to recall that one hundred years ago Bright recognized that in certain forms of the disease which bears his name the blood was deficient in protein and suggested even at that time that albuminuria was the cause of oedema. According to Moore and Van Slyke, when the total plasma protein falls from the normal level of 6 to 8 grams % to 5.5 grams %, oedema is likely to occur. This, of course, is due to the decrease in colloid osmotic pressure exerted by the diminished plasma proteins. The most important protein in this regard is the albumen fraction, since it forms approximately 60% of the total plasma proteins, and exerts, according to Govaert, four times as much osmotic pressure as the globulin fraction.

Just as several factors enter into the mechanism of oedema, so several types of oedema may be recognized. It is well to keep these types in mind, and when confronted with an oedematous patient, an effort should be made to ascertain the exact cause of the swelling so that proper treatment may be instituted. The following modification of Christian's classification I have found helpful:

TYPES OF OEDEMA:

1. Circulatory Failure Oedema—(a) Cardiac, (b) Hepatic.
2. Renal Oedema—(a) of Acute Nephritis, (b) of Renal Protein loss.
3. Nutritional Oedema—(a) Due to Plasma Protein Deficiency. (1) Diarrhoea, (2) Starvation, (3) Unbalanced Diet. (b) Due to Abnormality of Plasma Protein Formation.
4. Anaemic Oedema.

5. Inflammatory Oedema.
6. Anaphylactic Oedema.
7. Obstructional Oedema—(a) Venous, (b) Lymphatic.
8. Endocrinal Oedema—(a) Myxoedema, (b) Associated with Menstruation.

The most common type of oedema is that due to circulatory failure of cardiac origin. When a patient presents a picture of generalized oedema, one should first suspect failure of the right heart with engorgement in the systemic venous circulation. The history of such a case is usually typical: gradual increasing shortness of breath, fatigue, palpitation, cough, and swelling of the ankles which is more noticeable at night. The clinical history is substantiated by physical examination. The heart is usually enlarged; valvular disease may be present or absent. There is congestion at the lung bases and the liver is often enlarged. Hydrothorax and ascites may be present. The oedema responds to the influence of gravity. Involvement of the face is rare.

Cardiac oedema responds to any treatment which tends to improve the output of the right heart. Moderate or large accumulations of fluid in either pleural cavity should be drained as an initial measure. This procedure, alone, frequently initiates diuresis probably through improvement in the general circulation. Prolonged rest is essential. Digitalis in the form of pills or capsules of the powdered leaf should be used in adequate dosage. It is advisable to limit the fluid intake and impose a salt-free diet. Salt substitutes and lemon juice may render the diet more palatable. When oedema in congestive heart failure is not relieved by the above regime, it usually responds to the diuretic action of certain drugs; members of the purine group such as theobromine, theophylline or diuretin are effective and are but slightly toxic. Where more vigorous diuretics are required the newer mercurial compounds, either salyrgan or mercupurin, are of inestimable value. It is advisable to use an acid salt, preferably ammonium chloride, for two or three days as a preliminary measure. Care must be used to avoid toxic effects, since an occasional patient is highly sensitive to mercury. A test dose of .5 cc. should be given intravenously. If no unpleasant reactions occur, the dose may be increased to 1 or 2 cc., being cautious that none of the drug escapes into the surrounding tissues. The diuretic may be repeated once or twice weekly as required. The introduction of these mercurial diuretics represents a noteworthy advance in the treatment of cardiac failure. They have modified the course of the closing stages of chronic heart disease, so that now it is possible to rid the water-logged tissues of fluid and restore the condition of the patient to one of comparative comfort.

One form of cardiac failure with oedema frequently gives rise to confusion and that is the type of failure associated with a normal rhythm and a slow heart rate. Very often, the patient gives no history referable to the heart, oedema or swelling of the abdomen being the first complaint. Unless care is taken the enlarged heart may be overlooked. As

a result of chronic passive congestion of the kidneys, the urine is scant, is of high specific gravity and contains much albumen and some casts. Because of the urinary findings, a mistaken diagnosis of nephritis may be made. If ascites predominates and the liver is enlarged, cirrhosis, or even malignancy may be suspected. If this type of failure is kept in mind, there should be no mistake. Where doubt exists, therapeutic measures directed at heart failure produce a dramatic response. It is well to keep in mind another type of heart disease that is often masked as hepatic, because of the associated recurrent ascites. This disease was described by Pick as an extreme grade of chronic venous engorgement of the liver in cardiac patients with adherent pericardium. The clinical features are: recurrent ascites, enlarged firm liver, absent or slight jaundice, and no oedema of the legs. The patient usually gives a history of rheumatic fever. The heart may be enlarged but murmurs are not conspicuous. Broadbent's sign, if present, may give a clue to the diagnosis. Fluoroscopic examination is extremely valuable. The recognition of this syndrome, rare though it be, is important, since surgery offers great relief through stripping off the thick adherent pericardium.

Circulatory failure of hepatic origin as it occurs in various forms of cirrhosis of the liver is readily recognized. The history of epigastric discomfort, inappetence, morning nausea, retching and vomiting, is fairly typical. Examination reveals a characteristic cirrhotic facies, and the large abdomen contrasts with the attenuated frame. The liver and spleen are usually enlarged and in many cases there is evidence of a collateral compensatory circulation. It is well to remember that oedema of the lower limbs may precede ascites. The formation of ascitic fluid is due primarily to obstruction of the portal circulation. In cases of long standing, hypo-proteinaemia associated with malnutrition and loss of protein from the blood into the ascitic fluid is a contributing factor. Oedema of hepatic origin responds poorly to treatment. Ordinary diuretics are useless, although salyrgan combined with ammonium chloride may induce moderate diuresis. These drugs fail, because the mechanism by which absorption of fluid takes place is inadequate in cirrhosis to allow for a continued satisfactory response. With failure of diuretics, abdominal paracentesis should be performed, and repeated as required. In certain cases, surgical interference with omentopexy may be worth while.

Oedema of renal origin is the second most common cause of generalized oedema. It occurs in two forms: (1) Due to acute nephritis, (2) Due to protein loss in the urine associated with chronic glomerulonephritis or nephrosis—if one chooses to designate the latter as a clinical entity. The diagnosis of renal oedema is relatively simple. The distribution of the swelling and study of the urine directs attention to the kidney.

Oedema in the early stages of acute nephritis is probably the result of diffuse capillary damage. In more protracted cases, a fall in the colloid osmotic pressure, due to leakage of protein through the damaged

vessel wall, is an additional factor. In rare cases of acute nephritis, oedema may be enhanced by increased hydrostatic pressure secondary to a failing myocardium. In the vast majority of cases, oedema is slight in degree and of short duration, requiring no treatment.

The second type of renal oedema, that due to albumen loss in the urine, is associated with subacute or chronic renal disease. Whether or not nephrosis exists as a distinct clinical and pathological entity is beyond the province of this discussion. With all available evidence, it seems to me more satisfactory to consider it as a phase in the cycle of glomerulonephritis. Regardless of opinion, all meet on common ground in recognizing a definite clinical picture which has been termed through custom or for want of a better name, *"the nephrotic syndrome."* It is characterized by: massive oedema with fluid in the serous sacs, massive albuminuria, oliguria, waxy pallor, good renal function, typical urinary sediment, low total plasma protein with reversal of the albumen globulin ratio, increased blood cholesterol and low basal metabolic rate. Certain findings are noteworthy because of their absence, namely: hematuria, elevated blood pressure, arteriosclerosis of the retinal or peripheral vessels, cardiac hypertrophy, renal failure and true uraemia. The course is typically chronic, with remissions and exacerbations. The patient's appearance is often deceptive, oedema obscuring the underlying malnutrition, which is an important feature since it predisposes to intercurrent infections; the pneumococcus is the chief offender. In rare instances, apparent recovery may occur after weeks or months of hope and despair. The vast majority of cases, however, sooner or later progress into the terminal stage of glomerulonephritis. The long continued albuminuria is the important factor, resulting in a depletion of the plasma proteins with reversal of the albumen-globulin ratio. Theoretically, management should aim at preventing the leak of albumen through the glomerular tufts, and replacement of the diminished plasma proteins. To alter the permeability of the glomeruli is at present beyond our power, so that treatment resolves itself into building up the deficient plasma protein and ridding the body of oedema. A diet rich in protein is urgently indicated, although the patient, water-logged as he is, may find great difficulty in handling 150 to 200 grams of protein daily as advocated by Epstein. In the average case, 100 grams of protein is sufficient for a diet yielding 2,200 calories. Most patients prefer protein in the form of meat. To those who find the diet burdensome, a goodly portion of protein may be administered in the form of edible casein in doses of 20 to 30 grams in a glass of milk. A high protein diet may yield dramatic results, though often it is disappointing. Salt should be withdrawn or restricted to not more than 3 grams. Fluids should be restricted, but not to the point of making the patient uncomfortable. In purely renal oedema, water is retained only with enough salt to make a physiological saline solution. Salt restriction is the important factor. In fact, water drinking may induce diuresis by washing out the salt. In patients who fail to respond to the above regimen, the use of diuretics

should be considered. The purine and saline group are useless. Urea in large doses of 50 to 75 grams a day, given in lemonade, is worth a trial. Diuresis usually takes several days to appear, is moderate in degree and ceases when the drug is discontinued. The mercurial compounds, either salyrgan or mercupurin combined with ammonium chloride, are more effective than any other means at our command in ridding the body of superfluous fluid, and if used cautiously can do no harm to the kidney.

Nutritional oedema has been described as "war oedema," "prison dropsy," "hunger swelling" and "deficiency oedema." It was prevalent in Europe during the World War, when many people were forced to exist on a diet consisting largely of fluids, deficient in protein and rich in salt. Hypoproteinaemia, as in the nephrotic syndrome, forms the underlying basis for this type of oedema. It is usually due to a deficiency of protein in the diet, but may result from improper digestion or absorption of protein from the intestinal tract as occurs in certain cases of protracted diarrhoea. Precipitating factors include—excess ingestion of water, excess salt, and incipient heart failure. Treatment consists in feeding sufficient protein to replace the previously wasted body protein.

Cases of idiopathic hypoproteinaemia form an interesting group. They resemble in type, renal or nutritional oedema, except for the fact that albuminuria is lacking and there is no evidence of protein deficiency in the diet. Not long ago a case of this type came to my attention—a middle-aged woman whose only complaint was generalized oedema. Physical examination, except for oedema, was entirely negative. Repeated examinations of the urine failed to reveal albumen. Blood studies showed a low total plasma protein, with a low albumen fraction and a relatively high globulin fraction. Similar cases have been reported by Christian, Cope and Goodby, Myers and Taylor. The exact explanation for the hypoproteinaemia in these patients is not known. It is probably due to abnormality in the formation of the plasma proteins.

Oedema is occasionally associated with severe grades of anaemia. The explanation of the oedema is rather obscure, for it is not attended by any consistent alteration of the plasma proteins. It is possible that some characteristic of the condition of anaemia tends to facilitate transudation. Increased capillary permeability, secondary to deficient oxygenation, may be the underlying cause, with accessory factors such as water intake, salt intake and early heart failure playing an important part. Treatment is directed at restoration of the blood to normal values.

Inflammatory and anaphylactic oedemas are usually readily recognized. Injury to the vessel wall allowing the escape of fluid and protein into the tissue spaces is largely responsible. Treatment is directed at the underlying cause.

Venous or lymphatic obstruction should be considered as a possible cause for oedema. This type of oedema usually involves the lower extremities, occasionally the arms. In thrombosis involving the large pelvic veins, the lower extremities may be greatly swollen. Patients

who suffer from varicose veins are subject to swelling of the limbs, particularly in warm weather or after standing for a considerable period of time. Obstruction to the thoracic duct may account for hydrothorax and ascites. If so, the fluid is of a chylous nature. Treatment of obstructional oedemas is chiefly surgical.

With a wider knowledge of the endocrine glands, we realize more and more the important role they play in various bodily functions. That they exert an influence on fluid exchange between the blood and tissues may well be possible. We are all familiar with the peculiar non-pitting oedema associated with advanced myxoedema, in which the interstitial tissues are flooded with mucoprotein. The response to adequate thyroid therapy is dramatic.

Recently, I saw a girl of 20 because of moderate generalized oedema occurring only with menstruation. Because of her swollen ankles, she had been told that she had heart disease. Examination was entirely negative, except for a moderate degree of oedema involving particularly the lower extremities. This case is similar to those reported by Thomas and Sweeny. Thomas reports that an anterior pituitary-like sex hormone controlled the swelling in his cases. This type of oedema illustrates the influence of the endocrines. Its mechanism is obscure, but it is probably due to some endocrine dysfunction or disturbance in the sympathetic nervous system.

SUMMARY

Oedema is a physical sign indicating excess fluid in the tissue spaces. Its mechanism is complex; certain factors are clearly understood, others remain obscure. An effort should be made to classify patients with oedema into various types. By so doing, appropriate therapy may be instituted with gratifying results.

WHAT EVERY WOMAN DOESN'T KNOW — HOW TO GIVE COD LIVER OIL

Some authorities recommend that cod liver oil be given in the morning and at bedtime when the stomach is empty, while others prefer to give it after meals in order not to retard gastric secretion. If the mother will place the very young baby on her lap and hold the child's mouth open by gently pressing the cheeks together between her thumb and fingers while she administers the oil, all of it will be taken. The infant soon becomes accustomed to taking the oil without having its mouth held open. It is most important that the mother administer the oil in a matter-of-fact manner, without apology or expression of sympathy.

If given cold, cod liver oil has little taste, for the cold tends to paralyze momentarily the gustatory nerves. As any "taste" is largely a metallic one from the silver or silver-plated spoon (particularly if the plating is worn), a glass spoon has an advantage.

On account of its higher potency in Vitamins A and D, Mead's Cod Liver Oil Fortified with Percomorph Liver Oil may be given in one-third the ordinary cod liver oil dosage, and is particularly desirable in cases of fat intolerance.

The Pathology of the Appendix*

By JOHN A. LEWIS, B.A., M.D.
Hamilton General Hospital, Hamilton, Ont.

THE appendix is a hollow muscular organ, situated at what was originally the apex of the coecum. It may occupy any one of five positions, viz.: behind the coecum, overhanging the pelvic brim, below and parallel to the coecum, and anterior or posterior to the terminal ileum. The appendix may be located by following one of the taenia coli, which fade out as they reach the base of the appendix. Great variations occur in the size of the normal appendix, and these can give rise to symptoms. Thus, when diseased, it may by contact, give sympathetic inflammation in such organs as the ureter, ovary or Fallopian tube.

Histologically, the appendix simulates large bowel. It has an external serous investment and a mesenteriole, which carries the appendicular artery. The longitudinal muscle fibres are not arranged in three bands, as in the colon, but form a continuous layer. The circular muscle envelopes the lumen at all but the hiatus Musenland. The sub-mucosa is relatively broad, and contains many well-developed lymph follicles; it is for this reason that the appendix has received the cognomen *"abdominal tonsil."* The mucosa is composed of columnar epithelium, which sinks into crypts, but forms no villi. The function of the appendix is shrouded in mystery, and most texts of physiology choose to ignore the question.

The pathological processes which may affect the appendix may be classified as:

(1) Inflammation—acute or chronic appendicitis.
(2) Appendicular obstruction.
(3) Neoplasms.

ACUTE APPENDICITIS

It is generally conceded that the source of infection in acute appendicitis is from within the lumen, but in a personally observed case at Victoria Hospital, London, identical strains of hemolytic streptococcus were isolated from the throat and from the appendix, thus indicating—a hematogenous spread. The infection enters through a break in the mucous membrane, usually at the base of a crypt. Polymorphonuclear leucocytes mobilize at the site, and depending on the dose and virulence of the invading agent, several terminations of the process are possible.

Resolution or fibrosis may occur and these are usually accompanied by a peritoneal plastic reaction, with the formation of adhesions. Unhappy cases may end with massive necrosis, perforation and peritonitis. Where healing and fibrous organization have been the finale, susceptibility to recurrence is markedly increased. Other sequelae may be a stenotic obstruction of the lumen of an empty appendix, resulting in a mucocoele, or an appendix containing feces of high protein content, ending in the obstructive type of appendicitis to be described later.

The acute process begins with lymphoid hyperplasia, polymorpho-

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nuclear infiltration of the submucosa, and the production of an acute inflammatory exudate on the mucosa. Vascular engorgement and cellular infiltration spreads peripherally, to involve the visceral peritoneum. The latter, by irritation, produces an inflammatory reaction in the neighboring parietal peritoneum, which is detected by the patient as the rather diffuse epigastric pain of early acute appendicitis. This is a referred type of pain felt in the somatic skin areas supplied by the afferent nerves serving the peritoneum.

CHRONIC APPENDICITIS

The diagnosis of chronic appendicitis has been the subject of much debate, the pathologist inclining to be skeptical, the surgeon indulgent. The presence of fibrosis alone can hardly be a criterion, as many appendices, removed during the course of other operations, and apparently asymptomatic, may show a fibrotic process. Eosinophilic infiltration peripheral to the sub-mucosa (in which it is considered to be normal) is considered by some a sure indication of this rather vague pathological entity. Fatty deposits in the submucosa represent the "tombstones" of previous inflammation. Boyd believes sub-peritoneal lymphatic congestion and lymphocytic accumulations to be significant signs of "chronic appendicitis." A milky opacity, or loss of lustre of the serosa is the criterion for many, but periappendicitis produces the same picture. These, however, are points of academic interest, and the important and difficult question which always confronts the surgeon at operation is: Can this appendix give rise to the symptoms present?

The acutely inflammed, or gangrenous appendix is obvious, even to the novice. The question is not so easily answered in other cases. When the appendix found is a rigid fibrotic tube, the possibility of recurrence is rather good. The shrunken, obliterated appendix would scarcely seem to be a source of symptoms, unless through associated adhesions. Such a finding should certainly indicate further exploration. A lustreless serosa, obscuring the finer vasculature and containing dilated larger vessels indicates at least a mild degree of active disease.

On section, the "chronic" appendix usually presents a dilated lumen, flattened folds of mucosa, attenuation of all the layers but the serosa, and a loss of well-marked distinction between the sub-mucosa and the muscularis. In more fibrotic appendices, the whole wall may present a hard, white, uniform fibrotic appearance. In such specimens, the lumen may be dilated, or may be entirely obliterated.

Boyd would seem to have supplied the best solution to the problem. "The pathologist is frequently consulted as to whether a given appendix could produce the symptoms complained of. . . . It appears to the writer that no pathologist is in a position to return a satisfactory answer. . . . The man to settle the matter is a surgeon with a good pathological training who will follow the subsequent history of his private cases for a number of years."

APPENDICULAR OBSTRUCTION

Willsie attributes a large proportion of the preventable mortality

of appendicitis to an obstructive lesion, giving rise to necrosis and gangrene distal to the obstruction. The appendix is, in the final analysis, a loop of bowel closed at one end. Impaction of inspissated fecal matter, very rarely with a foreign body as its nucleus, may occlude the lumen. Bacterial proliferation and trophic vascular disturbances occur distally. Ulceration of the mucosa takes place about the fecolith, the appendiceal wall becomes attenuated, infected, and rapidly undergoes a gangrenous change. The lumen is filled with foul-smelling, hemhorragic fecal matter. In this type of disease, the omentum acts less promptly, and while the visceral peritoneal reaction is quick enough, it is hampered by the subjacent trophic change. Perforation, here, much less frequently meets an organized peritoneal response.

Homans recognizes this lesion, but Dean Lewis (in Cecil's Medicine), quotes Aschoff's opinion that the foreign body actually tends to protect the mucosa. A limited experience in the pathological laboratory has convinced the writer that such lesions can and do occur. They are characterized by an indefinite clinical picture, differing from that of the classical acute inflammatory lesion. Torsion of the appendiceal mesenteriole, or thrombosis of the appendiceal artery, may give rise to a similar pathological picture.

As previously stated, foreign bodies may form a nucleus for a fecal concretion, though the days of the fashionable "cherry-pit" appendicitis are past. A definite percentage of pathological appendices, particularly in children, contain Oxyuris vermicularis; these have provided the irritation necessary to incite inflammatory symptoms. The various forms of Ascaris may also be found.

NEOPLASMS

The only tumour of comparatively common occurrence in the appendix is the "carcinoid" tumour, present in .3-.6% of cases. It is believed by Masson to be a paraganglioma (derived from the cells of Kulchitzki and Schmidt, as in the glands of Lieberkuhn). This tumour usually forms a small bulbous swelling at the tip of a fibrous appendix. On section, it appears as a fairly uniform, light yellow layer, encircling the lumen. The histology is not typically adenomatous, the cells arising primarily in the sub-mucosa and infiltrating the muscularis. The cells are spheroidal, displaying the staining reaction with silver which is common to all argentaffine tumours.

These tumours are benign and do not metastasize. Adenocarcinoma of the appendix is a rather rare tumour, but does occur.

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Gratitude is humbly expressed to Dr. J. H. Fisher for precept and permission to cite the case at Victoria Hospital, and to Dr. Geo. Ramsay, who supplied the physiological concept.

Harvey and His Work*

By H. O. FOUCAR, M.D.
London, Ontario

IN thinking of Harvey I have been trying to place him correctly in relation to other individuals and events of his time. We know that he was born in Folkstone in 1578 and that he died in 1657 at the age of 79. He was born, therefore, during the reign of Queen Elizabeth (1558-1603) and lived through the reigns of James I (1603-1625 and Charles I (1625-1649) and the period of the Commonwealth (1649-1660); but he died before the accession of Charles II (1660-1685). He travelled throughout England, Scotland, Italy and Germany. During this period many stirring events happened, some of which touched Harvey closely, for he was associated directly or indirectly with all the crowned heads of England in his time. In 1605, he married Elizabeth Browne, the daughter of Queen Elizabeth's physician. His brother John was attached to the court of James I and it may have been through him that he was able to bring the King's letters in support of his application for appointment in 1609 as assistant physician to St. Bartholomew's hospital. Later, he was appointed physician to James I and finally became physician and close friend of Charles I. To him was entrusted the care of the royal children, later Charles II and James II, during the battle of Edgehill in 1642.

One of the events which must have thrilled his boyish heart was the defeat in 1588 of the mighty Spanish Armada under Philip II. This occurred in the English Channel, almost at his doorstep, and altered the history of Europe. Spain lost her power and the Netherlands gained their independence. Throughout Europe we witness bitter religious strife between the Catholics and Protestants, culminating in the Huguenot Wars (1562-1629) in France and the Thirty Years War in Germany (1618-1648). In England we find evidence of this same conflict. Henry VIII had broken away from the Roman Catholic Church and established the Church of England. He confiscated the property of the monasteries and Parliament passed a bill called "an Act for the Abolishing of Diversity of Opinion." Under Edward VI the Catholics were persecuted while, on the accession of Mary, the tables were turned and in five years between two and three hundred Protestants were executed. Many of these were burned at Smithfield just outside the confines of St. Bartholomew's hospital. Today a plaque may be seen on the walls of this hospital commemorating these martyrs.

Harvey witnessed the struggle between James I and the members of the House of Commons over the idea of Kingship. Charles I fell heir

*An address delivered at the Seventeenth Annual Banquet of the Harvey Club, London, February, 1935.

to both fights, the civil and the religious. He had the same notion of the divine right of kings and this brought him into conflict with the House of Parliament, resulting eventually in civil war in England. Harvey accompanied Charles I in 1639 on his trip to Edinburgh, when he tried to impose the English liturgy upon the Scotch Presbyterians and so started trouble in the North. As a direct result of these two mistakes, Charles was tried and executed in Whitehall on January 30, 1649.

During his absence, Harvey's rooms in Whitehall were ransacked by the Ironsides and many of his scientific notes and specimens were destroyed. It is significant that, in spite of his close association with the King throughout both campaigns, he was not disturbed personally.

Harvey studied medicine in Padua from 1599-1603, returning to England, where he took his degree at Cambridge. In 1609 he received an appointment at St. Bartholomew's hospital, where he was expected to be in attendance one day a week. In 1632 a deputy was appointed because Harvey was absent so frequently in attendance upon the King. In 1643 a motion was made that Dr. Micklethwaite be appointed in the place of Dr. Harvey "who hath withdrawn himself from his charge and is retired to the party in arms against the Parliament." No action, however, was taken until ten years later, when Dr. Micklethwaite was elected. We see then that Harvey was associated with St. Bartholomew's hospital for 44 years, although not actively for the last twenty years. He was also engaged in private practice, but this fell off materially after he published his researches, although he still enjoyed one of the wealthiest and most distinguished practices in London.

All his life Harvey was collecting specimens, dissecting and making observations. His real claim to fame is that he applied the principle of observation and experiment to Medicine.

It is interesting here to speculate on the influence which Francis Bacon may have had on Harvey. Whatever criticism may be levelled at Bacon as a man or a politician, there is no gainsaying his position as a scholar and a philosopher. He insisted on experiment and careful observation of facts as the only true means of arriving at knowledge of the laws of Nature. We see that Harvey was one of the outstanding exponents of this principle. Doubtless they often discussed their respective problems for Harvey was Bacon's medical advisor. Mentioning Bacon reminds us that Shakespeare was also a contemporary.

We may well ask what were the general customs and mode of living of his day. Those who have read Pepys' famous diary will get an insight into the life of the time. It is true that Harvey antedated him by a few years but this discrepancy in time, however, is not very serious

and, as we read Pepys' experiences and reactions we can imagine Harvey living and working under similar conditions.

In studying his life I began to wonder how much of Harvey's London remains today. Harvey would have no difficulty in finding his way, although he might be bewildered by the traffic, over-awed by the buildings and surprised at the growth of Greater London. It is true that most of the city was destroyed by the fire of 1666 and that much of the remainder has given way to commercial expansion; still he would recognize the names at least of the main thoroughfares and even those of many of the buildings, although he would not know them in their present form, for they have been renovated, added to or completely rebuilt. Comparatively few structures exist now which were present in Harvey's day. In Holborn, he would find some Elizabethan houses, with projecting timered fronts which have resisted the march of time and stand as mute witnesses of an almost forgotten past.

We can easily imagine him wandering from Westminster and Whitehall to St. Bartholomew's, a route that he must have taken often enough during his lifetime. He would recognize Westminster Abbey easily for it existed then in its present form. We can see him strolling through the beautiful private chapel of Henry VII and then looking at the Royal Tombs. He would notice evidence of the vandalism of Cromwell's followers, the empty niches, the chipped figures and the damaged traceries and this would bring up sad memories. He would stop at the tomb of Elizabeth and would notice that Essex's ring has at last been returned to its original owner. He knew the story and would be interested in the sequel. From here he would cross the street to the Houses of Parliament. They are, however, of very recent origin. His attention would doubtless be drawn to St. Stephen's Hall, occupying the site of the old St. Stephen's Chapel, where the Commons met for years, and to Westminster Hall, where his patron Charles I stood for trial.

It would be natural for him to wend his way along Whitehall, where at one time he lived. All seems different until his eyes fall on a building to the right, now the Royal United Service Museum, containing national trophies ranging from a Saxon shield to relics of the Great War. This was designed by Inigo Jones, a personal friend of Harvey, as Whitehall Banqueting Hall and was to be part of a new palace which was never completed. For Harvey this building doubtless would recall many happy memories, but they are eclipsed by one tragedy, for he cannot forget that it was up those stairs and out of that window that his patron Charles I stepped onto a specially erected platform to be beheaded.

Trafalgar Square, with the Admiralty Arch, Nelson's Column and the lions with the National Gallery in the background are all new, but he would recognize St. Martin's-in-the-Fields, although the fields have

long since disappeared. Unconsciously he would turn to the right along the Strand. One familiar object catches his eye—Charing Cross. Not the original nor even in the original site, it represents the last of the Eleanor Crosses raised by a grateful mouring King, Edward I, to mark the places where his wife's coffin had rested as it was carried from York to Westminster.

The Strand has changed tremendously. It used to be the river road. All that remains now to remind him of the fine estates which formerly lined the Thames are the names of the cross streets. Instead of the old river front with its shelving mud bank he would see the Thames Embankment, a modern improvement. Probably the only building on the Strand that he would know is Somerset House, now used as Government offices.

I do not know whether Harvey was an antiquarian but we might find him turning down Strand Lane just to see whether the old Roman Bath still remained. Gone is all its ancient splendour. The beautiful tiles and mosaic work which lined it, before its owner was called back to defend Rome against the barbarians, have been stripped off, leaving only an ugly shell. It is still fed by the cold springs from Highgate, miles away, but it is now no longer in a stately country villa but it situated in a back alley with the ugly pipes of London's waterworks running over it.

As Harvey nears the City of London proper his steps would quicken and he would look about expectantly. The old Temple Bar, the barrier or entrance to the city, is gone, condemned as an obstruction to traffic. In its place is a monument surmounted by the Griffin. He cannot resist the temptation to turn to the right through a narrow lane and finds himself in the Inns of Court, the home of the lawyers from time immemorial. This section shows less change probably than any other. The Temple Church, founded by the Knight Templars in 1185, later given to the Knight Hospitallers (about 1300) and finally bought by the lawyers of the Inner and Middle Temple, still stands and has recently been restored. It is one of the best examples of Norman and Early English styles. The Temple Gardens still exist and Harvey might well remember Shakespeare's description of the scene which is said to have been the origin of the Wars of the Roses. According to tradition, in this garden, in 1430, the followers of York and Lancaster plucked white or red roses to denote their respective allegiance. Continuing along Fleet Street, he would turn north up Old Bailey. Newgate Prison is gone but on the left he would see the Chapel of St. Sepulchre, where he was married. This would also bring him to St. Bartholomew's hospital, the scene of his labours for so many years. This is the oldest hospital in London and was founded by Rahere in 1123 in connection with the Priory Chapel of St. Bartholomew the Great, which exists

close by. Haphazard teaching of Anatomy was commenced in 1734 but lectures were not really organized until 1763 under Percival Pott, over a hundred years after Harvey's death. Only recently have the cloisters been demolished to make room for new buildings. Harvey would probably have as great difficulty finding his way around as we do, for there is very little left of the old part.

In this vicinity may be seen St. John's Gate and the Priory Church of St. John, founded in 1504 by Prior Docwra of the Order of the Knights of St. John of Jerusalem, now the headquarters of the St. John's Ambulance Association. Near here, too, is Charterhouse, founded by the Carthusian monks in 1341. Part of the London Guildhall was not destroyed by fire and further to the east the Tower of London stands today much as it did in Harvey's time.

With few exceptions these would be the only buildings of importance to link us with the London that Harvey knew.

I have made no attempt to discuss Harvey's scientific contributions for his work on circulation and on generation is familiar to you all and needs no eulogy from me. Patiently he plodded on, dissecting, experimenting, observing, testing each link in his chain of evidence. He was in no hurry to publish, for he wished to be sure of every point. From his work, he was satisfied to conclude that blood flowed as if in a circle. He was never able actually to visualise the entire circle but he was convinced that it existed and that there was no other way of explaining the phenomena he had observed and he had the courage to state his convictions. The demonstration of the capillaries microscopically supplied the missing link and placed Harvey forever among the immortals.

HARVEY'S PRIVATE LIFE

Dr. Wm. Harvey came to London in July, 1646, and lived with his brother Eliab, on Dowgate-Hill. Oughtred's "*Clavis Mathematica*" was always on his study table. Aubrey, a gossip-monger of the time, informs us that he kept a pretty servant girl to wait on him, and states, "he no doubt made use of her for warmth." He took care of this girl in his will, which adds some corroboration to Aubrey's story.

In studying, he advised to go to the fountain-head at once, and read Aristotle, Cicero and Avicenna; the new authors, he called "shit-breeches." He wrote a bad hand. After his book on The Circulation of the Blood came out, he fell off greatly in his practise, being supposed by the people to be mad.

When he had the gout he would sit with his legs bare, even if it were frost, on the leads of Cockaine-house. He would even put them into a pail of cold water till he was almost dead with cold, then betake himself to his stove, and so it was gone. This cooling practise, mentioned by Pliny, has been attempted to be revived in the present day.

—*From* "PROFESSIONAL ANECDOTES," 1825.

A Case of Aleukemic Lymphatic Leukemia

By ROWE FRY, B.A., '37

MISS E. P., aged 16 years, was admitted to the Memorial Hospital, St. Thomas, on May 17th, 1936. She complained of tiredness and was noticed to be paler than usual.

Previous Illnesses: The patient had had measles and mumps in childhood, and had undergone a tonsillectomy in 1930.

Family History: Her father and mother were both alive and well. She had no sisters or brothers.

History of the Present Illness: Four or five weeks before admission, she first noticed that she seemed rather paler than usual. The patient then began to complain of an increasing feeling of tiredness, and fainted several times while at school. She was given tablets containing iron, but her condition did not improve. A week before admission she noticed an unusual shortness of breath on climbing the stairs. Following this, she vomited and vomited almost every day until admission. Her menstrual periods had always been normal, but the last two were rather short and there was less flow than usual.

Physical Examination: On examination, one was struck at once by the extreme pallor of the patient. There was some enlargement of the spleen. A haemic murmur was heard over the apex of the heart. The patient complained of a sensation of numbness in the extremities. No enlargement of the lymph glands was noticed. The temperature and pulse were normal. Her blood picture was as follows:

Hb — 15%; RBC's — 1,600,000 per cu. mm.; C.I. — 0.4; WBC's — 3,100 per cu. mm.

Differential:	Total PMN's - - -	28%
	Mature forms - -	12%
	Young forms - -	16%
	Total Lymphocytes - -	72%
	Large cells - - -	12%
	Small cells - - -	60%

The red cells were microcytic, hypochromic, pale ring forms. No immature red cells were observed.

The vision was slightly impaired. Ophthalmoscopic examination showed oedema of the retina, with retinal haemorrhage and very pale areas between the haemorrhagic spots.

The bleeding time was 35 minutes and the clotting time was 6 minutes. No clot retraction had occurred after 4 hours.

In view of the severe anaemia and the leukopenia, a tentative diagnosis of aplastic anaemia was made.

Clinical Course: The patient had a daily rise of temperature which

varied from 101° to 104° F. Her appetite was poor and she vomited occasionally. A transfusion of 500 cc. of whole blood was given, following which the haemoglobin rose to 32 per cent. She felt better for two or three days but soon relapsed to her previous state. Iron in various forms was administered. Three more transfusions, totalling 1,100 cc., were given over a period of eleven days, but these afforded only temporary relief.

On June 1st, 1936, following her third transfusion, the blood picture was as follows:

Hb—21%; RBC's—1,490,000 per cu. mm.; WBC's—1,100 per cu. mm.

Differential:		
	Total Polymorphs - -	16%
	Young forms - -	8%
	Mature forms - -	8%
	Lymphocytes - - -	84%

The anaemia gradually increased until on June 4th, before the fourth and final transfusion, her haemoglobin was 11% and RBC's 1,040,000. The patient died on June 7th, 1936, with a temperature of 106° F., 22 days after admission to the hospital.

Post-Mortem Findings: The bone marrow of the long bones and ribs appeared red and glistening, rather uncharacteristic of aplastic anaemia, in which disease the marrow is fatty and atrophic. Microscopically, it was almost entirely replaced with closely packed lymphocytes. There was no hyperplasia of the myeloid elements of either the granular or red blood cell series.

The spleen was enlarged and weighed 28 ounces. The splenic pulp was congested and contained numerous lymphocytes.

The sigmoid colon showed purpuric spots in the mucosa.

The portal areas of the liver and the interstitial areas of both the cortex and medulla of the kidney were packed with lymphocytes.

The stroma of the ovary showed numerous lymphatic deposits.

The structure of the lymph glands was disorganized. No germinal centres remained and lymphocytes were scattered diffusely throughout the reticulum.

Besides the lymphatic deposits mentioned above, there were other changes in the tissues, referable to the severe grade of anaemis. The liver and kidney showed cloudy swelling and the myocardium exhibited cloudy swelling and fatty degeneration. There was evidence of increased blood destruction, as shown by the hemosiderin pigmentation of the reticulo-endothelial cells of the spleen and liver.

SUMMARY

Due to the acute fulminating course, high fever, chills, headaches and prostration, this case was undoubtedly one of acute lymphatic leukemia in the aleukemic phase. There was no lymphatic hyperplasia, but that is quite consistent with such a diagnosis.

The interesting features of the case were the severity of the anaemia and the fact that the patient died in the aleukemic phase of

her illness. It seems reasonable to suppose that this patient had always been in the aleukemic state, since the beginning of the disease.

So fulminating was the condition that time was not permitted for this state to end and for the lymphocytes to pass into the blood stream and give the typical picture of lymphatic leukemia.

Two other facts are demonstrated by this case:

(1) Blood transfusions and iron are futile, except as palliative measures in the treatment.

(2) In leukemia, there need not necessarily be a leukocytosis, but there is always a change in the differential count.

DR. HERMAN BOERHAAVE

This celebrated physician and medical professor was originally intended for the clerical profession, and his brother for the medical. A singular accident occasioned their respective pursuits to be counter-changed.

Some works of Benedict Spinoza having been published at Leyden, the criticisms on the Hebrew text contained in it were the general subject of conversation among the learned men and students in the universities; the Christian clergy were loud in their condemnation of the learned Jew. Boerhaave happened, at this time, to be one day returning from an adjacent town to Leyden in a common passage-boat, or "treickschuyt," and one of his passengers was declaiming, with great violence, against Spinoza and his atheistical tenets.

Perceiving Boerhaave to wear the dress of a divinity student, he addressed himself particularly to him, expecting to hear an echo of his own sentiments. But Boerhaave had already discovered that this violent disclaimer was, in fact, talking beyond his knowledge; and instead of joining him in his abuse of Spinoza, calmly asked him, "If he had read his works?" to which the stranger replied, "He had not, and should esteem it wicked to look even into them." "How, then," returned Boerhaave, "can you pretend to judge them?"

This mild retort silenced the stranger; but it was reported through the whole of Leyden, in the course of the next day, that Boerhaave had turned Spinozist. In consequence of this slander he found it would be vain to seek promotion in the church, and, accordingly, he and his brother exchanged their professional studies.

—*From* "PROFESSIONAL ANECDOTES," 1825.

An Organic Foreign Body in the Tracheobronchial Tree

By W. J. TILLMAN, M.D., F.R.C.P.(C.)
London, Ontario

FOREIGN bodies in the tracheobronchial tree are fortunately not very common. The organic bodies most frequently found are peanuts, and the roasted ones are looked upon as the most irritating. One sees, at times after operation, pieces of tonsils and adenoids and teeth lodged in this area. Among inorganic substances, metal objects are the most common.

When a child is suddenly taken with an attack of suffocation and syanosis, and a cough resembling croup, the physician should always think of a foreign body in the respiratory passages, even when the history would not suggest it. A foreign body, like a glass bead or pea, may travel up and down the trachea, lodge in the larynx and cause a repetition of these attacks.

Chevalier Jackson says that the diagnosis of an organic foreign body in the tracheobronchial tree can be made from the following points:

1. History.
2. Dyspnoea and restlessness.
3. Toxemia and occasional delirium.
4. Dusky cyanosis or intense pallor.
5. Distressing cough with pinkish gray, thick tenacious sputum.
6. Irregular septic type of temperature.

He also states that the severity of the symptoms is in reverse ratio to the age of the patient.

Case History—Baby W., aged 11 months, while being fed bacon for her breakfast on the morning of December 20th, 1936, commenced to laugh. She started to cough at once, as though choking. The bacon seemed to go down alright, but she brought up a little froth. In about ten minutes wheezing began, but the baby otherwise seemed perfectly well and played around and ate her meals all day. At night she became restless and would not sleep. She had some vomiting, and the wheezing became very much worse.

The patient was first seen by the writer the next day, Monday, at 3 p.m. Examination revealed very marked asthmatoid breathing and marked bronchitis over both lungs. There was impaired air entry on the right side, whistling rales were present in great numbers; the pulse was fast, and movement was decreased on the right side. I advised an immediate X-ray, which was taken by Dr. Wismer.

X-Ray Report—Fluoroscopic examination of the chest reveals an asthmatic type of breathing on both sides. The lung fields appear clear and the heart is not displaced. There is no foreign body shadow. There is, however, a decided possibility of the presence of some non-opaque

foreign body, most likely located in the right lung root. This conclusion is based on the following points: (1) Very early atelectasis along the right heart border, (2) Irregularity of the right leaf of the diaphragm, (3) Some infiltration around the middle lobe bronchus.

Ordinary asthmatic remedies gave no relief, and the asthmatic wheezing became very much worse. The child did not rest at all that night, and when I saw it at noon the next day, temperature had risen to 102 degrees, breathing was labored and asthmatic, and the pulse was fast. I advised the parents to take the child at once to Dr. William A. Hudson in Detroit, for bronchoscopic examination.

Dr. Hudson's report is as follows:

X-ray studies previous to the removal of the object showed that the right lung was very much distended with air, and the heart, together with the mediastinum, was shifted to the left entirely. The left border of the heart was snugly against the left chest wall. There was very little difference in the inflation of the lung during inspiration or expiration.

Repetition of the X-ray studies and physical examination gave ample evidence of an obstructive lesion of the bivalve character, involving the right stem bronchus. There was marked obstructive emphysema of the entire right lung, with compression atelectasis of a considerable portion of the left lung. The temperature was 102 degrees by rectum. The physical signs entirely agreed with the X-ray findings. Examination previous to bronchoscopy revealed decreased breath sounds over the left hemi-thorax, with scattered rales. On the right, during the first half of the inspiratory effort, no breath sounds were heard; during the last half of the inspiratory effort a bronchial type of breath sound was present. Bronchoscopy was performed at 8 p.m. on December 22nd. At 8 a.m. the temperature was 102 degrees, reflecting the maximum reaction due to the instrumentation.

The tracheobronchial tree was explored with a 4 mm. bronchoscope. A mass was found filling the right stem bronchus, which permitted a very slight passage between itself and the walls of the bronchus on full inspiration. This mass was grasped with a side grasping forcep and a considerable portion of it was removed. Some of the same material was projecting into the middle lobe bronchus. This was grasped with the smallest side-grasping forcep and removed entirely. The patient's air hunger, which had previously very much distressed her, was relieved immediately. Before the instruments were removed from the patient's trachea she had relaxed and adjusted herself for a good sleep. The material removed had all the appearance of a well-cooked piece of meat rind.

The patient's rectal temperature at noon the next day was 100.2. Physical findings were within normal limits, except for a few scattered rales. The patient was allowed to return to London on December 24th, 1936.

This case exemplifies very clearly one of the acute conditions that

may follow the entry of a foreign body into the lung. On the other hand, a child may have a foreign body in its lung without producing any symptoms. There are cases on record where a child has had a peanut in its lung for one year, without symptoms, and has then coughed up the peanut with no further trouble. This is not the ordinary history, however. The foreign body may set up a septic pneumonia which is later followed by an abscess formation and the patient often dies as a result, from toxemia.

ON CONVALESCENCE

"To be sick is to enjoy monarchial prerogatives. Compare the silent tread and quiet ministry, almost by the eye only, with which he is served—with the careless demeanour, the unceremonious goings in and out (slapping of doors, or leaving them open) of the very same attendants, when he is getting a little better—and you will confess that from the bed of sickness (throne, let me rather call it) to the elbow chair of convalescence, is a fall from dignity, amounting to a deposition.

"How convalescence shrinks a man back to his pristine stature! Where is now the space, which he occupied so lately, in his own, in his family's eye?

"The scene of his regalities, his sick-room, which was his presence-chamber, where he lay and acted his despotic fancies—how is it reduced to a common bedroom! The trimness of the very bed has something petty and unmeaning about it. It is *made* every day. How unlike to the wavy, many-furrowed, oceanic surface, which it presented so short a time since, when to *make* it was a service not to be thought of at oftener than three or four-day revolutions, when the patient was with pain and grief to be lifted for a little while out of it, to submit to the encroachments of unwelcome neatness, and decencies which his shaken frame deprecated; then to be lifted into it again, for another three or four days respite, to flounder it out of shape again, while every fresh furrow was an historic record of some shifting posture, some uneasy turning, some seeking for a little ease; and the shrunken skin scarce told a truer story than the crumpled cover-lid.

"Hushed are those mysterious sighs—those groans—so much more awful, while we knew not from what caverns of vast hidden suffering they proceeded. The Lernean pangs are quenched. The riddle of sickness is solved; and Philoctetes has become an ordinary personage.

"Perhaps some relic of the sick man's dream of greatness survives in the still lingering visitations of the medical attendant. But how is he, too, changed with everything else? Can this be he—this man of news—of chat—of anecdote—of everything but physic—can this be he, who so lately came between the patient and his cruel enemy, as on some solemn embassy from Nature, erecting herself into some high mediating party? Pshaw! 'tis some old woman."

—*From* "THE CONVALESCENT," *by Charles Lamb.*

Dr. Elam Stimson*

By EDWIN SEABORN, M.D., F.A.C.S.
London, Ontario

THE more remote ancestry of the subject of this sketch were English, and can be traced back several centuries. Many families of the name, sprung from a common stock, are to be found in England. His immediate ancestry were all natives of New England. To study and practice medicine seems to have been a family trait, so many Stimsons in tolerably regular succession having been doctors. James Stimson, M.D., an eminent practitioner of Hartford, Connecticut, was great-grandfather to this Elam Stimson. The one of whom we write, the youngest of a family of twelve children, was born in Tolland, Connecticut, October 4, 1792. When he was about ten years of age his parents suffered reverses of fortune, and until they died, thirteen years after, his strong filial affection was shown by his untiring and successful effort (seconding that of his next older brother, Joel) to provide for their maintenance.

* * *

"Dr. Elam Stimson served in the United States army during the War of 1812, first as a substitute for a drafted man, for the term of three months, at New London, Connecticut; then he enlisted for one year, the greater portion of which time was passed on Staten Island, N.Y. He was third sergeant of his company."

* * *

"For his services in that war he received from the United States Government, about thirty years later, a bounty of one hundred and sixty acres of land, which he located in the prairie country of Northern Indiana."

* * *

"At the expiration of his term of enlistment he returned to his home and commenced the study of medicine. To obtain necessary means, he laboured on a farm or taught school. A friend who owned a cranberry marsh gave him permission to make what he could from one year's crop. He hired help to pick the berries, marketed them in Hartford, and netted one hundred dollars by the operation. That hundred dollars, he remarked later in life, seemed to him the greatest financial lift he ever had.

"By dint of persevering effort, he gradually accumulated enough money to meet the expense of a course of lectures. Meantime, while labouring or teaching, he had been reading medicine under the direction of Dr. Thompson, of Tolland.

"His first course of lectures was at Yale College, New Haven,

*This outline of the life of Dr. Elam Stimson consists in part of direct quotations from various authorities, which are acknowledged as they appear. It will serve as an informative introduction to the "Cholera Beacon," which follows it.

Connecticut. The tickets read curiously, as compared with those of today, and give one a good idea of the comparatively narrow scope of medical teaching in the early part of our century, even at one of the then best seats of learning in America. There were but four lecturers—a 'Corporal's guard' as compared with the 'full staff' of lecturers and teachers attached to each of the principal medical schools of today."

* * *

"The lecturers were: On Chemistry and Pharmacy, B. Stillman; Anatomy and Physiology, J. Knight; Theory and Practice of Medicine, Surgery and Midwifery, Nathaniel Smith; Materia Medica and Diseases of Children, Eli Ives.

"The summer following this course of lectures was spent in the office of an eminent medical man of Hartford, Connecticut, Mason F. Cogswell, M.D.

"His next course of lectures was at Dartmouth, Hanover, New Hampshire. 'Chymical' lectures were by J. T. Dana, and lectures on 'Practice of Physick' and on 'Obstetricks,' by R. D. Mussey, M.D. At Dartmouth he graduated Valedictorian of his class, August 18, 1819."

* * *

"While yet an undergraduate, he married, January 10, 1819, the eldest daughter of the Rev. Augustus Bolles, a man prominent in the Baptist Church, of superior ability as a preacher and editor."

* * *

"He returned from Dartmouth to his native place and engaged in the practice of his profession. An influential man in that community afterwards said to him: 'You have served me faithfully and well in three different capacities, as a farm servant, as a teacher, and as my family physician.' "

* * *

"Prospects of financial success not being brilliant in the East, with the pioneering enterprise characteristic of his ancestry he removed, with his family, to the then 'far West,' and settled in St. Catharines, Upper Canada, in the spring of 1823. St. Catharines was then but a hamlet, on the 'Twelve Mile Creek.'

"Learning it was necessary he should be examined by a Government Board of Examiners and receive a license from the Governor before he could legally practice in Canada, he crossed, in a schooner, from the mouth of the Niagara River to York (now Toronto), and presented himself for examination before 'C. Widmer, Grant Powell and R. C. Horne, Esquires,' who expressed themselves as particularly well pleased with his proficiency, especially in anatomy—that of the brain. July 7, 1823, he was licensed to practice by Lieut.-Governor Sir Peregrine Maitland, of the Province of Upper Canada."

* * *

"Some time in 1824 he removed to Galt. His practice there was very extensive, Paris, Princeton, Drumbo, Ayr, Hamburg, Waterloo, Berlin, Guelph, Preston, East and West Flamborough, St. George, all

are within the then sparsely settled country over which he travelled on horseback with capacious saddle-bags, green-baize leggings and heavy dark 'surtout,' ministering to the needs of the sick, poor and rich, oftener very poor than even moderately well-to-do, for those were pioneer days. Sometimes he was away from home for days together, sending word home where those needing him could find him."

* * *

"In the latter part of 1828 he removed still farther west, to London, and continued the practice of his profession. Here his ride was as extensive and his labours as arduous as in Galt.

"Among his documents is the following on heavy, gilt-edge foolscap:

"GOVERNMENT HOUSE, May 30, 1831.

"SIR,—I am directed by the Governor to acquaint you that a commission has been prepared appointing you a Coroner for the London District, and is now ready for delivering to any agent whom you may authorize to receive it.

"I have the honour to be, Sir,

"Your most obedient, humble servant,

"Z. MUDGE, Secretary.

"DR. ELAM STIMSON."

The foregoing information was derived from Canniff's Medical Profession, Upper Canada.

* * *

Dr. Campbell in "Pioneer Days in London" says:

"A better known teacher of this period was Miss Stimson. She was a daughter of one of our early physicians—Dr. Elam Stimson. She was a well-educated lady, and with the aid of her niece, Miss Grannis, conducted a very successful school for the short time she remained in London. The building and equipment was not very elaborate. Her first schoolhouse was a log building of one room, while the furniture consisted of a few benches for the scholars and a desk and chair for the teacher. Later she moved into their own building on the corner of Ridout and Carling Streets. It is said that when some of her boys misbehaved they were sent down cellar; but as the imprisoned lads found it convenient to get at the jam jars she had to adopt some other means of punishment."

* * *

In a paper read before the London and Middlesex Historical Society, January 16, 1917, Dr. Campbell states that Dr. Stimson opened an office on Ridout Street, just north of Dundas Street.

In material found in the Tower Room of the County Court House of London by the writer, in 1936, the proceedings of the Court of Quarter Sessions were brought to light. This material had largely to do with the Cholera Epidemic of 1832. A few extracts only will be made:

"London District Dr.
To Elam Stimson.

			£	s	d
May	7, 1831	—Call & med for Indian	0	3	9
		7 boxes Blue oint	0	1	3
	17,	Vt Indian open Tumor & Empl deach	0	5	0
		Vt Ward and Cath	0	2	6
		Vt Murray Going v.s. Emet & Cath	0	5	0
	18,	Vts Murray & Indian & drugs Allen	0	8	9
	19,	Vts Murray, Indian & med	0	8	9
	20,	Vts Murray, Indian & med	0	8	9
	21,	Vts Murray, Indian & med	0	8	9
	23,	Vts Murray, Indian & med	0	8	9
	26,	Vts Murray, Ward & med	0	8	9
June	1,	Vt Indian Ward & med	0	8	9
	4,	Vt Murray Indian & med	0	8	9
	5,	Vt Murray Indian & med	0	8	9
	6,	Vt Murray & med	0	5	9
	7,	Vt Murray & open Tumor	0	5	0
	12,	Vt Murray Cath—E E for Indian	0	5	0
	14,	Vt Indian & 3 Cath powders	0	5	0
	15,	Vt Empl deach pills &c	0	5	0
	17,	Vt. Murray & med	0	3	9
	23,	Vt E. E. for Indian Emet & Sal E	0	5	0
July	5,	Vt Stevenson in jail & v.s	0	3	9
	6,	Vt Stevenson & oint for Ward	0	5	0
Aug.	4,	Vt Murray & Tonic bitters	0	3	9
	11,	Vt Indian & med	0	5	0
	14,	Vt Indian & med	0	5	0
	16,	Vt. & med for Indian	0	3	9
Sept.	9,	Pills—Blue Oint & Sal Epsum for Sealy & Brady	0	2	6
			£8	7	6

Approved in open Genl. Qr Sssns of the
Peace at London 14 April, 1832.

M. BURWELL
Chairman."

Also the following:
"(1) Account presented to the Court of Quarter Sessions,
April, 1833.

(N.B.—I have only given a few of the items on this account. The total Amount comes to £20-10-0.—E.S.)

To Elam Stimson (Surgeon)

			£	s	d
May	29, 1932	—Inquisition upon the body of Catherine Edwards	2	10	0
		Dissection	1	5	0
		Constable's fees	0	15	0
June	10,	Gum Opium (Sundry times)	0	5	0
Oct.	9,	Attendance upon prisoners, up to April, in jail	7	10	0
Sessions—1833					
		Attendance & Chloride of lime to fumigate Court House during Assizes	1	5	0

And as follows:

Sept. 5, 1832

BOARD OF HEALTH; London Dr.

To Bemis Pixley.

		£	s	d
Bemis Pixley	To two yards of Cotton Bot	0	1	3
	To one Sheet for shroud for Jones	0	5	0
	Do. to one Cotton Sheet destroyed in use	0	5	0
	Do. paid for Cleaning Room, Cleansing &c	0	5	0
	Do. To Boarding Coloured men and nurses	0	7	6
	To Washing of Bed Close used with Peter Rogers & Coloured man while sick	0	5	0
	Do. To three Bottles of Brandy used in sickness with Jones, Rogers & Pulin	0	7	6
	To one sash & Glass Broken by Peter Rogers while sick	0	5	0

To Shirts and Pantaloons used for sick.....	0	5	0
Do. To Trouble of House House room & other necessary trouble in time of sickness.....	1	15	0
	£3	11	3

August 30th, 1832.

BEMIS PIXLEY

I certify that the above account is in my opinion correct and reasonable.

ELAM STIMSON, Srgn.

London, Sept. 5, 1832.

Recd payt from Wm. Robertson the above amount.

BEMIS PIXLEY"

Again from Canniff's Medical Profession, Upper Canada, 1894:

"During the prevalence of an epidemic of Asiatic cholera, his wife fell a victim to that 'scourge of Nations,' July 20, 1832, having been but twelve hours sick. Five days after, their youngest child, of two years, died of the same disease, and a daughter also was so low with it that she was robed in her grave-clothes ready to be coffined. But she regained consciousness and was restored to health. At his wife's death he had five children, the eldest of whom was aged but little more than thirteen years."

* * *

In September, 1832, he was instrumental in organizing the London District Medical Association, meetings of which were held at Joe Loder's Inn at Otter Creek.

Again from Canniff:

"Dr. Stimson visited Hartford, Conn., and married Susan Bolles, his deceased wife's sister. Returned to Canada, he concluded to depart from the scene of so great affliction, so he wound up his affairs in London and settled late in August, 1833, at St. George, a pretty village—the centre of one of the most beautiful and fertile portions of Canada. Here, with the exception of a time spent in practice in St. Catharines, to afford better educational facilities for his children, he continued to reside and practise . . ."

In 1835 he published the "Cholera Beacon." A copy was discovered in the Surgeon General's Library at Washington. This pamphlet follows. His death occurred in 1869. His monument is to be found at St. George, Ontario.

SURGEON DROWNED

A surgeon aboard a ship-of-war used to prescribe salt-water for his patients in all disorders. Having sailed one evening on a party of pleasure, he happened by some mischance to be drowned. The captain, who had not heard of the disaster, asked one of the tars, next day, if he had heard anything of the doctor. "Yes," answered Jack, after a turn of his quid, "he was drowned last night in his *medicine chest!*"

"The Cholera Beacon"*

By ELAM STIMSON, M.D.

TO THE READER

The Author presents this brief and humble publication to the Public unaccompanied by any apology. To the humane, intelligent, and liberal of the profession, and public generally, the importance and necessity of more particular and general knowledge of the incipient stage of Cholera is too apparent for the Author to anticipate any charge of vanity, or affected superiority.

No general assertion is more true than that—"Cholera is easily cured if taken in time," and yet thousands of its victims have remained unalarmed while the disease was making its covert and fatal approaches, and with physicians at their very doors. No advantage can be derived from the trite admonition to "Apply in time," so long as the patient is in ignorance when that time is. Neither can the hasty and desultory precepts tendered during the prevalence of the disease prove of general utility. Highly excited fears pervert the judgment of many, and render them more fit and probable subjects of attack, while erroneous ideas of the first symptoms, and too great reliance on their own judgment, subject others to the greater danger of passing unalarmed that stage of the disease in which proper medical aid can be of any avail.

The object of "The Cholera Beacon" is to remedy these evils—to allay unfounded fears—to eradicate erroneous impressions, and in their place, to substitute that knowledge of the disease and its appropriate treatment, as will enable the reader to avert a fatal attack. In short, our object is to give timely warning of approaching danger.

It was not consistent with the brevity and general design of this essay to enter into a more particular detail of facts alluded to, nor to enlarge upon the arguments arising from them.

We have imbibed our theory from an attentive perusal of the Book of Nature—from a contemplation of the whole phenomenon of the disease; and to the same source of information we would refer the profession, rather than to any ingenuity in the arrangement of facts or arguments in this paper.

We have not undertaken the task of guiding popular practice in

EDITOR'S NOTE:—The Journal takes a particular pride in offering to its readers this interesting and literary document. Through its pages, one can feel the terror of a pioneer people before a savage plague, whose tragic onslaughts they were helpless to control. One cannot help sympathizing with the medical men of the day, whose naive speculations on the etiology and treatment of cholera are adequate testimony of their bewildered efforts to stamp out a disease which decimated whole settlements and took their friends and relatives from them, one by one.

The only known copy of this pamphlet in existence is now at the Library of the Surgeon General in Washington, U.S.A. Dr. Seaborn, an ardent historian, has had a photostatic copy of the original made for his own use, and we are very grateful for his kindness in allowing us to make this publication available to our readers. Due to its length, it will be presented in three instalments, of which this is first. Second and third will appear in February and April numbers of Journal.

Cholera, unmindful of its importance—neither from a confidence of superior qualification, but from the want of some prospect of a similar publication from a more able pen, and from the fullest confidence of the correctness of the general principles, and superior efficacy of the remedies recommended.

During the laborious and unremitting practice we have not been able to prepare these sheets for the press in a manner satisfactory even to ourselves; but that it will be the means of greatly diminishing the mortality of the disease, and amount of domestic afflictions, which we, in common with so many of our fellow-men, have shared, is the sincere wish of

THE AUTHOR

St. George's Village, Dumfries, May, 1835.

CHOLERA literally signifies "bile flux"—when applied to the disease of which we are about to speak, none could be more inapplicable, because the bile in the course of the disease is suppressed. But from its having been so universally known by the term "Cholera" we must consent still to retain it—but only as a name, and on condition that the reader will fully divest his mind of any preconceived opinion, of its being a disease of the abdomen.

We should now be understood that all our remarks, and precautions, are intended to apply to persons occupying situations where the Cholera is, or is expected to become epidemic; or to persons that have been exposed to its epidemic influence.

It is supposed that covering a vast extent of country—perhaps surrounding the world, an impure state of the atmosphere exists, tending to produce Cholera. This may be called general infection. In certain situations local causes operate to increase this contaminated state of the atmosphere; and this may be called local infection. To the union of these we apply the term Epidemic influence. Of the cause of the general infection we pretend to know nothing—but it would seem that the local infection is the product of heat and humidity, holding in solution a quantity of miasm, of exhalations of decaying animal or vegetable matter. Hence we find Cholera has prevailed most in the vicinity of great water courses, and in low and marshy situations.

The inland town of London, U.C., the place of our residence in 1832, stands upon a peninsula formed by the junction of two main branches of the Thames. Here the disease had apparently a spontaneous origin, and prevailed with great virulence and fatality.

The general infection we consider insufficient to render the disease prevalent—but producing sporadic or scattering cases in persons who happen to be in a certain state of ill health, or having a high degree of susceptibility.

The most striking and melancholy example within our knowledge of the generation and effects of the local infection occurred in this vicinity in the summer of 1834.

On the 28th of July, 1834, Galt, a village on the Grand River, U.C., was visited by showmen with a menagerie. It was exhibited under an awning of canvas, nearly enclosed at the sides, and drawn together in a conical form almost to the top. The day was excessively warm, and the crowd suffocating. The exhibition lasted about three hours. It is estimated that about 10,000 persons were present, and that not less than 200 persons died of Cholera within ten days. The population from which the assembly at the exhibition was composed, in the townships in the vicinity of Galt, is supposed to be about seven thousand.

The first case was in one of the showmen, who sickened on that day, which was Monday. No other case occurred until the following Wednesday morning—on that day not less than thirty were attacked, all of whom had been at the show. The greatest number of cases were on the Thursday and Friday following—but new cases occurred for several days. In speaking of an attack, we here allude to the time the patient supposed the attack commenced—the time he was "taken down." The average length of time the disease lasted after this event was about sixteen hours.

Four days previous to the exhibition of animals at Galt, two children of Mr. J. G., on the Governor's Road, 12 miles southeast of Galt, were attacked with Cholera, one of which died. On the same day (24th July) two cases of what we shall call second-grade Cholera came under our care, being the first that occurred of that form of the disease within our knowledge that season. About this time also, many were affected with first-grade symptoms—but with the exception of the children alluded to we have not been able to learn that any case of fully developed Cholera occurred in this part of the province previous to the exhibition of animals at Galt; and for several days subsequent to that event, and in which more than two hundred were attacked with Cholera, all had been at that exhibition with only two or three exceptions. From the 6th of August the disease became more general and was not confined to such as were at the menagerie. About this time it appeared at Hamilton and Dundas—situations more low and marshy than Galt, and adjacent to Burlington Bay, or the head of Lake Ontario. From these facts it is evident that a deteriorated state of the atmosphere existed previous to the 28th of July, yet the fatal catastrophe following the exhibition at Galt was mainly attributable to the highly vitiated, or imperfectly oxygenated air, produced by the numerous and sweltering crowd under the canvas—the ventilation being altogether inadequate for so numerous and crowded an assemblage. It appears that at Hamilton, Dundas and several other situations the epidemic influence was the product of the more common causes of general infection, united with a local infection, which last is caused by the action of heat upon putrescent vegetable matter. These few remarks are sufficient for the reader to anticipate our answer to the oft-repeated and naturally interesting question—"Do you think Cholera contagious or catching?"

A contagious disease we would define as one that is produced by a

specific virus or morbid matter, that has either by contact or in the form of sweat—vapour from the breath—or some other excretion from the body, emanated from the sick of the disease, and which is capable of producing the same disease in another person. According to this definition, Cholera is not contagious. But it is not denied that the excretions or other filth incident to a sick room, or any other cause rendering the air more unfit for respiration may have the effect to render the epidemic influence more efficient. From the above facts and observations, that danger which is real may readily be distinguished from that which we consider imaginary.

Among other causes tending to impair the purity of the atmosphere are some which ignorance and credulity have brought in general use as preventives of the disease.

The smoke and fumes of burning tar can have no other effect when inhaled than to render the process, and benefit to be derived from respiration less perfect.

Chloride of lime, and some other substances have the reputation (undeservedly in our opinion) of destroying or of rendering the epidemic influence inert. Their operation at least must be very limited, for the air in a room or house, if at all ventilated, is displaced by the slightest current.

Some have imagined that a quantity of infection may become attached to the floor, walls, or furniture of a house, as it sometimes does in contagious diseases like the smallpox. Although we never entertained any fears of Cholera from this cause, yet all houses where this or any other disease has prevailed (and occasionally at other times) should be well cleansed by general ablutions and whitewashing.

OF THE FIRST GRADE OF CHOLERA

During the prevalence of Cholera (and generally for some time previous to its appearance), in any particular town, village, or section of country, unusual morbid sensations are experienced by many persons, inhabiting such situations, which have commonly been called "premonitory symptoms." Many, if not most that are affected with these never have the disease fully developed at all—while others that have but few of these symptoms, and these few so slight and transient as scarcely to be noticed, have serious and even fatal attacks—still these symptoms are produced by the same cause as Cholera in its aggravated form. They ought then to be called first grade of Cholera.

The symptoms of first grade of Cholera are the following: A faint, fluttering or trembling sensation at the heart, headache, dizziness, ringing or buzzing in the ears, cramps, generally of the calves of the legs, which occur most frequently in the night, an aching and numb sensation in the limbs, often shifting to different parts of the body, sharp pains, loss of appetite, indigestion, sickness at the stomach, an uneasy, full sensation of the abdomen, or heavy moving pains and irregularity of the bowels, an oppression of the chest, giving rise to frequent sighing, or to an inclination to make a more full and deep inspiration (a longer

and deeper breath), than a common sigh, a sense of weariness, and exhaustion upon using slight, or but ordinary exercise, and if the exercise be continued often produces a faintness or fluttering of the heart, a tight, oppressed or heavy sensation at the pit of the stomach, a sense of creeping coldness on the surface of the body, and sometimes short and "hot flashes" of fever—the mouth and tongue have sometimes a more soft and slippery appearance than is natural—and also of a more dark and dusky color.

This lengthy catalogue of morbid sensations applies to their appearance in a considerable number of cases collectively. They seldom, we may say never, all occur in the same individual—indeed it is not common for but few of these symptoms to affect the same person.

TREATMENT OF FIRST GRADE OF CHOLERA: It is not always necessary to have recourse to medicine for this grade of Cholera; but if the symptoms are severe, and especially if there is distress, or a heavy oppressive sensation at the pit of the stomach—or if there is a heavy faint or fluttering sensation at the heart, it will be advisable to bleed a pint or more from an adult; and if the bowels are not regular give 15 or 20 grs. of Calomel. After this it is often necessary to give some strengthening medicines, as a grain of quinine two or three times a day—or, what we think preferable, Huxham's Tincture (see Appendix) in teaspoonful doses three or four times a day. If there are wandering, or cholic-like pains in the bowels, give a dose (two or three teaspoonfuls) of Elixr. Pro. once in six or eight hours. It will allay the pains and afterwards operate as physic, producing bilious stools. If, after a time, the symptoms return, recur again to the same treatment.

Caution.—The tight, oppressive, or heavy sensation at the pit of the stomach—often called "a load at the stomach"—the loss of appetite, or some other symptoms, sometimes induce persons to take an emetic. This should be avoided—neither should any nauseating or cathartic medicines be given, except Calomel, Elixr. Pro., Hiera Picra, or some other warming physic.

OF THE SECOND GRADE OF CHOLERA

The grade of disease we are now to describe generally affects children—but sometimes youths, and less frequently adults. It prevailed extensively, and proved fatal to many in the neighborhood of London, U.C., at the time Cholera prevailed in '32. It was much more extensive than Cholera in its worst form, which was almost entirely confined to the town, while the milder form of Cholera covered a considerable extent of country. It was also prevalent during the time of Cholera in this vicinity (Dumfries) in '34—but within our practice it proved fatal but in a single case.

The symptoms are—irregularity of the bowels, and often wandering cholic-like pains. The evacuations from the bowels are mostly mucous, of the color and consistence of thick cream—and the mucous often intimately mixed with black blood in various proportions—some black blood alone is discharged, or mixed with but a small proportion of

mucous. The tongue has a soft, smooth, moist, or greasy appearance, which as well as the lips and inside of the mouth, all of a darker hue than natural—or all of a dark leaden color—sometimes the tongue is covered with a coat of exceeding smooth, short and thick fur, which is of a brownish color, and it is always moist. The patient has commonly much thirst, though sometimes it is quite moderate. The surface of the body is for the most part of the time cool, or colder than natural; but there is commonly some irregular paroxysms of fever that last only two or three hours, often not so long. For some time previous to the setting in of this grade, some of the first grade or premonitory symptoms are present, such as a loss of appetite, indigestion, irregularity of the bowels, and the weak, fluttering sensation at the heart. Both the severity and duration of this second grade (when uninfluenced by medicine) is liable to great variations in different patients—some cases terminate fatally within two or three days after they are thought to be seriously ill—others linger eight or ten days and often recover.

TREATMENT OF THE SECOND GRADE OF CHOLERA: If the disease is but slight, give to a child from 3 to 7 years old (and others in proportion to their age) 2 teaspoonfuls of the Elixr. Pro. and, if necessary, repeat in 5 or 6 hours, for two or three times. It will almost always allay the pains or uneasy sensations in the bowels, and afterwards produce bilious stools. These should be followed by strengthening medicines, such as Huxham's Tincture in teaspoonful doses, 2 or 3 times a day, and if the state of the bowels require, i.e., if they continue out of order, alternate the use of Huxham's Tincture with the Elixr. Pro. for physic.

But if the disease is more severe, and there are discharges of whitish or cream-colored mucous from the bowels, and the patient mostly cold, having only "hot flashes" of fever—or if the lips and tongue have a more dead and dark appearance, or if there is sickness at the stomach, and much purging of any kind, efficient means should be resorted to. One or two small bleedings will be of great service; but whether this be used or not, Calomel should be given in doses of 8 or 10 grains, once an hour, until two or three doses are given. If the skin, as well as the lips and tongue, are cold, or if the last is quite dark or purple, 2 or 3 grains of Capsicum should be given with each dose of the Calomel, and hot ginger tea may be given between these doses. After the last dose of Calomel has been given about 3 hours, if it does not operate, follow it with a teaspoonful of Elixr. Pro., and repeat this dose once an hour until it operates as physic. Dry heat (by warming flannels) should be applied to the surface of the body. In many cases the above must be repeated once in a day or two for some time. Most commonly something like a regular continued fever comes on after the operation of the physic, which in bad cases is a good sign. If there should not be much regular or continued fever, some strengthening medicines should be given for several days, even while it is necessary to give the Calomel and other physic.

Caution.—Give no emetics or other sickening medicines—neither

any other physic than the kinds we have mentioned, or some other of a warming nature—nor opium in any form except there are severe pains in the bowels, when 8 or 10 drops of laudanum may be given and repeated after an hour if the pain continues.

OF THE THIRD GRADE OF CHOLERA

We come now to speak of the third grade or fully developed Cholera. It is the only grade of the disease that has usually been known by the name of Asiatic Cholera, or Cholera Asphyxia.

To guard the uninformed and unwary against its incipient, insidious and fatal attack is the principal design of this essay.

The reader must not rest with but a cursory perusal of these precautionary lines, but the different forms of attack must be attentively studied; otherwise while the mind reflects upon one form in which it apprehends the disease will approach, it assumes another, and thus imperceptibly and disguisedly secures its victim.

The variety of symptoms by which Cholera may be developed may be divided into four:

(1) Regular Attack: By commotion in the bowels and diarrhoea.
(2) Irregular Attack: By a diarrhoea of thin, light-colored or greyish stools.
(3) Constipated Attack: By a costive state of the bowels.
(4) Bilious Attack: By a bilious diarrhoea.

As these different forms of attack all converge in a watery diarrhoea, a separate description will be given of each up to that stage. The disease then assumes a dreadful uniformity, and a single description only will be necessary.

There is still another variety, which we have termed Primary Cardiac Congestive Cholera. As this does not run into the watery flux, it will be considered in another place.

The first variety or form of attack here described we have called a Regular Attack, because it is not complicated with any effort of nature to carry off the disease by a substituted secretion.

The commencement of a regular attack is by commotion in the bowels and diarrhoea.

The form of attack begins with a sense of rumbling or commotion in the bowels, often emphatically expressed by the patients saying their "insides were all in an uproar," or that it "seemed as if their bowels were all turning upside down." This commotion is usually unattended by pain. After some time, varying from thirty minutes to two or three hours, there is commonly a large discharge from the bowels of feculent matter, and some portions of food not fully digested. Within an hour or two this discharge is followed by another, which appears to empty the bowels, the stools being composed of fecal matter, chyme, and the food last taken partly digested. This also is attended with some pain—in some cases considerable, in others slight. One or both of these discharges is almost always very large. These have a feted smell. At the time, or soon after this second evacuation, there is commonly sickness

at the stomach, in some cases slight, in others it amounts to vomiting—or there is a sense of faintness, or fluttering at the heart—and often a general tremor of the whole system—sometimes these symptoms attend the first discharge from the bowels, but more frequently and severely the last.

There is now commonly an interval of several hours duration, in which no other symptoms are present but weakness, or a little faintness or fluttering at the heart, and sometimes a little sickness at the stomach. After this interval, which in different cases varies much, there is a sudden call to stool. It is now watery or very thin, and it passes from the body without effort and with a sudden gush. This is attended with very little or no pain. The less pain the more danger. This stool is soon followed by another, and another, which are now or soon after attended by vomiting, spasms, etc.

Sometimes this form of attack varies considerably from the above description—slight cramps of the toes, sickness at the stomach, and even vomiting sometimes precede the first purging. In other cases these symptoms occur after the first evacuations, and before the watery diarrhoea. Again, after one or two watery stools, the diarrhoea may cease without sickness at the stomach or vomiting, and return again after an interval of twelve, twenty-four, or even thirty or forty hours. Sometimes the emptying of the bowels (i.e., by the two first stools) may be performed at one evacuation, and in other cases by three or four and attended with much griping and pain.

Although in this form of attack as above described, little or no pain is experienced, yet there is sometimes an indescribable distress, through the whole body, and particularly at the pit of the stomach, and a sense of fullness or heavy aching of the head—some patients have spoken of this distress as attending the general tremor. The intensity of this distress varies in different cases from the most extreme suffering down to that which accords with the general description.

Of the second form, or Irregular Attack: This is by a diarrhoea of thin, light-colored or greyish stools. Although we have placed this grade second in the description, it is first in frequency of occurrence. As in the regular attack, this is commonly preceded by some of the first grade or premonitory symptoms—and particularly by nausea or sickness at the stomach. Many times there is the commotion in the bowels, but this is less distinct, and not so uniformly present as in the regular attack—but a loss of appetite more uniformly precedes this than the first form. If but little or no pain attends diarrhoea, the danger (as in the first form) is greater, and the nearer it approaches to the watery relax. On the contrary, if there is considerable pain and griping in the bowels, and the stools very feted, the immediate danger is somewhat less, and may be cured by less efficient means. But such cases often unexpectedly run into a watery stage—the pain, griping and fetor of the stools diminish suddenly—and vomiting, spasms, the watery flux, and other alarming symptoms come on almost at same time. In some few cases the vomiting and spasms have preceded the truly watery stools.

(To be continued)

Abstracts

PRONTOSIL

A Brief Summary From the Current Literature With One Case Report

THE merits of blood stream antiseptics are based on the fact that their molecular structure is of a more unstable nature than that of the cellular protoplasm. They are therefore introduced into the blood stream in the hope that the toxins present will combine more readily with them than with the cell protoplasm. Quinine is probably the oldest of these; mercurochrome of late has been used but no definite beneficial effects have been generally acknowledged.

Recently, much has been written on the remarkable results derived from *prontosil*. This is a definite organic compound (a member of the azo dyes) which is evidently of outstanding value in the treatment of *streptococcus hemolyticus* septicaemia. A review of the recent literature shows that it is of definite value in puerperal sepsis, erysipelas, lymphangitis, empyema, meningitis, due to *streptococcus hemolyticus*.

Evidence shows that this substance, on gaining the blood stream, is broken down to a sulphonamide, or some closely related substance, which is apparently definitely bacteriocidal to the *streptococcus hemolyticus*.[3] The chemical nature of prontosil is extensively discussed by Hoülein.[1]

The drug is available in two forms: *prontosil soluble* for intramuscular administration, and *prontosil album*, which is available in tablet form (0.3 gm.) for oral use. The two compounds are different chemically, and it appears that the latter form is more lethal to streptococci *in vitro* than the former. Colebrook, Buttle and O'Meara (1936)[2] have shown that *prontosil album* (sulphonamide) has a definitely inhibiting action on the bacteria *in vitro*; prontosil soluble has no such action. It is suggested by Trefauel and others (1935)[2] that the effective action of prontosil soluble is derived from its conversion to sulphonamide in the blood stream. Fuller has shown that sulphonamide is excreted when prontosil is given by mouth or vein.[2]

It is remarkable that there has been perfected a compound which is destructive to the virulent streptococcus hemolyticus when taken by mouth.

Foulis and Barr[3] report the merits of *prontosil album* which was administered to seventy patients with definite bacteriological evidence of either *streptococcus hemolyticus* septicaemica or peritonitis, resulting from puerperal sepsis. In this series there was one death (1.4%) as compared with a mortality of 13.5% previous to the administration of prontosil alba.

At St. Joseph's Hospital, London, Ontario, there was a child, J. M., aged 8 months, suffering from *streptococcus hemolyticus* septicaemia. This patient received both forms of this compound (¼ tablet of prontosil album and 1 cc. of prontosil soluble intramuscularly q4h). After four hours there was a temperature reduction from 105° to 100.2°. The temperature has been maintained at not above 99° since.[4] It is advisable to continue the drug two or three days after the fever drops. Experience has shown that the toxicity is low.[3]

The drug is a new one and has yet to stand the test of time. If all that is claimed of it be true, and it certainly appears to be, then the medical profession have been introduced to a blood stream germicide which is available in two forms and which possesses no rival.

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[2]Fuller, A. T., Lancet, No. IV., Vol. 1; Jan. 23, 1937; 194-198.
[3]Foulis and Barr; Brit. Med. J., Feb. 27, 1937; 445-446.
[4]Courtesy V. A. Callaghan, M.D., F.A.C.S., and H. J. Loughlin, M.D.

W. E. CRYSLER, '38,
Abstract Editor.

ERYTHEMA NODOSUM

By W. W. SPINK

Arch. Intern. Med.; 1:65,1937

Erythema Nodosum is a nodular rash, which occurs on the arms and legs as painful reddish blue nodules, the size varying from a millimetre to several centimeters in diameter. It appears to be a non-specific inflammatory reaction of the skin to a variety of bacterial, toxic and chemical agents. In a series of one hundred and thirty-three cases, the peak of incidence was in females, ages from twenty to twenty-nine years, and the greatest number of cases occurred in the early spring.

In the author's experience, the association of erythema nodosum with streptococcal infection has been much more common than with tuberculosis. A general review of the literature is presented and ten cases of the disease are studied critically.

R. LAWSON, '37

ACUTE HAEMATOGENOUS OSTEOMYELITES IN CHILDREN

By V. L. HART

Jour. Amer. Med. Ass.; 108; 7, P. 524, 2-13-37

This disease is a local manifestation of a generalized, transient haematogenous infection. The bone lesion is secondary to a remote infection. The most common infective agents are staphylococcus aureus, staphylococcus albus, streptococcus pyogenes and pneumococcus.

The disease first manifests itself as a metaphsitis or "metaphyseal furuncle." The metaphysis seems to be predisposed to the settling of bacteria because of the slowing of the blood current in the fine terminal capillary loops of the nutrient artery in this region, as well as the "paucity of phagocyte cells." The fact that the metaphyses are extremely vascular during the period of childhood and puberty seems to make acute haematogenous osteomyelites a disease especially of this period. The metaphysitis lasts for a variable period (generally twenty-four to forty-eight hours) depending upon the virulence of the invading organisms and the patient's resistence. Following this, the infection spreads by perforation of the "paper thin" cortical wall adjacent to the epiphseal plate and tracks subperiosteally along the diaphysis producing multiple areas of bone necrosis and invades the medulla through the Haversian canals. The relation of the joint capsule to the metaphysis (i.e. whether intra or extra capsular) largely determines whether the capsule will be affected. The infection may go farther and spread along intermuscular planes and into subcutaneous tissue.

During the earliest clinical phase the child is severely ill, complaining of severe and constant pain in the region of the joint or joints involved, although other signs of inflammation, synovitis and effusion, are absent. With perforation of the cortex these signs appear. Thus point tenderness during the metaphysitis is one of the most important clinical observations. Intense toxaemia, elevation of temperature and pulse rate, lencocytosis is also seen. History of trauma is helpful; X-ray at this stage is of no value.

If diagnosis is made which has been a rare occurrence during the acute metaphysitis surgical treatment may cure the disease, before widespread bone sequestration has taken place. A small incision is made directly over the part of the metaphysis that was extremely tender and the cortex in this region is exposed subperiosteally. Several drill holes or a 1" by ½" window is made in the cortex, the wound is lightly packed with petrolatum gauze (Orr method) and covered with sterile dressings and sheet wadding. A plaster cast is applied to prevent deformity and give rest to the part.

Operation is not necessarily followed by disappearance of clinical signs and symptoms of the acute infection since only a local manifestation of a general infection was treated here. Rest and fluid administration is the general treatment.

After perforation of the cortical wall and spread of the local disease process, the major issue is to improve the patient's general resistance. Then conservative local treatment, including incision, drainage, cast, is instituted. At this time the patient may pass into the chronic stage of the disease and the prognosis as to morbidity is appreciably altered.

GEORGE J. WEINSTEIN, '39

INTERPRETATION OF SOME COMMON DIGESTIVE SYMPTOMS

By J. W. SCOTT, Edmonton

Canad. Med. Ass. J.; 36; 1; Jan., 1937

It is important to elicit a full, accurate history, the order of development of the symptoms, and the degree to which they interfere with the patient's comfort.

A skillful interpretation of the following symptoms is of great diagnostic value:

Loss of Appetite—It occurs in the hypotonic stomach of generalized visceroptosis, pyloric stenosis with dilation, chronic gastritis and impaired tone due to neoplastic infiltration of the wall.

Loss of Weight—It is especially important after the age of forty. It is seen in nervous dyspepsia, but more commonly in duodenal ulcers and chronic gastritis due to excessive salivary secretion or to reflex closure of the cardia.

Heartburn—It results from functional dyspepsia, the over-use of tobacco and alcohol, cardiospasm, and less frequently in peptic ulcers.

Dysphagia—This is caused by disease of pharynx, pressure on the gullet from without, certain functional and organic nervous disorders, carcinoma of the oesophagus and cardia, and achylasia of the cardia.

Flatulence—Aerophagy causes the flatulence of nervous dyspepsia. It is common in duodenal ulcers, chronic gall-bladder disease, appendicular dyspepsia, and peptic ulcers with associated pylorospasm preventing passage of air into the pylorus for absorption.

Abdominal Discomfort and Pain—This is the most common and significant symptom in gastro-intestinal disease. We must consider the character, severity, location, radiation, duration, frequency, food relationship and periodicity of the pain. We are all familiar with the typical ulcer pain and the locations of referred pain. Pain that is severe enough to disturb sleep and to cause the patient to get out of bed for relief usually means organic disease.

If there is a change in characteristics of the pain of gastric ulcer, we must think of pyloric stenosis, hour-glass constriction, or malignant change.

Nausea and Vomiting—Nausea is seen in chronic gastritis and functional dyspepsia, but more often in extragastric disease.

Vomiting without abdominal pain usually indicates the absence of organic disease in the stomach. There is persistent vomiting in pyloric obstruction; occasional vomiting in uncomplicated gastric ulcer, gastric carcinoma without complete pyloric construction, and chronic gall-bladder disease. Vomiting gives relief from pain in gastric ulcers but little or no relief in carcinoma.

WALTER F. COPP, '38

CHRONIC OSTEOMYELITIS ASSOCIATED WITH MALIGNANCY

By HENDERSON AND SMART, Mayo Clinic
J. Bone J. and Surg.; 18; 56-60; Jan., 1936

Of 2,396 cases of chronic osteomyelitis at the Mayo Clinic, 5 cases or 0.302% showed malignant change. In these 5 cases draining sinuses of chronic ostesmyelitis had been present from 27 to 48 years. All cases were of lower extremity.

Symptoms—Swelling, increase in pain, haemorrhage, more perfuse and foul discharge.

Pathology—Epithelioma: 4 cases fibrosarcoma associated with squamous cell carcinoma in 1 case and no metastases in any case.

Etiology—Theories:

1. Continuous drainage of pus irritates skin.
2. Less efficient circulation in lower leg and in scarred areas.

Conclusions—

1. In cases of chronic osteomyelitis, every effort should be made to cause drainage of abscesses to heal by adequate treatment:
   (1) Orr treatment.
   (2) Maggot therapy.
2. Signs of malignancy in draining sinus: foul odor, pain, haemorrhage.
3. Treatment:
   (1) Confirm diagnosis by biopsy.
   (2) Amputation followed by deep irradiation.

PHILIP GLEASON, '37

TRAUMATIC ARTHRITIS

Arch. Surg; Vol. 33, No. 2; Aug., 1936; P. 213

Single severe injuries to the joints as well as mild repeated trauma are important factors in the etiology of arthritis. The lesion and the consequent irritation of joint structures and bone produce inflammatory reactions which lead to hypersecretion and hypertrophy of the membrane and to deformation of the articular surfaces.

Trauma not only presents the first link in the chain of pathologic changes in non-specific asteo-arthritis but may open a path for the invasion of the joint by non-specific organisma as well as by the tubercle bacillus and spirochaeta pallida.

The examination of joint effusions is an important means of determination of the severity of an injury and of differentiation between traumatic and inflammatory effusions and between specific and

non-specific conditions of joints. The aspiration of a joint evacuates pathologic products from the joint cavity and reduces irritation, thus promoting the restoration of normal conditions.

Requirements for the prevention of arthritis after injuries of joints or structures near joints are: reduction of fractures, early removal of loose or lacerated cartilage, and repair of ligaments. Proper therapy, with rest, fixation of the fracture and resumption of motion is of the utmost importance.

The early recognition of constitutional or acquired anomalies and diseases which are apt to traumatize or invade the joints, such as osteochondritis dissecous, Perthes' disease, genuvalgus and varus, syphilis, tuberculosis and endocrine disturbances, opens a way for preventative measure to reduce the development of chronic arthritis.

E. FAULDS, '37

FEVER THERAPH IN TABES DORSALIS

Relief of Gastric Crisis and Lightning Pains By the Use of the Kettering Hypertherm

By A. E. BENNET

J. Amer. Med. Ass.; 107; No. 11; 1936

The present therapeutic treatment for tabes dorsalis, particularly in the relief of resistant symptoms, such as lightning pains, gastric crisis, ataxia, bladder dysfunction, and paraesthesia are very discouraging.

Beside the method suggested in this article, two popular therapeutic measures are used at present for this condition:

1. *Chematotherapeutic Methods—*

These methods are used by the majority of practitioners:

(a) Courses of arsphenamine, neorsphenamine, mercury and bismuth salts with iodine therapy until there is a persistent negative spinal fluid and blood response.

(b) Treatment by arsphenamine, mercury and intravenous iodide followed by Swift Ellis injections.

(c) Tryparsamide.

(d) Bismuth arsphenamine sulphonate (Bismarsen).

Results—

Only about 50% of the patients show relief from resistant tabetic symptoms by this method.

2. *Malaria Therapy—*

The total percentage of improvement of resistant symptoms by this method is not much if any better than systematic routine chematotherapy. The malaria therapy works well in early stages of tabes, but gives poor relief in the late cases. In addition, the mortality rate of this therapy ranges from 10 to 20%.

3. *The Kettering Hypertherm plus Chematotherapy—*

The hypertherm is an air-conditioned cabinet in which the temperature can be regulated as desired. The technique in neurosyphilis is to give fifty hours of fever from 105° to 106° F. in ten sessions, combined with some form of Chematotherapy.

By this method the patient gets twice as much fever as obtained from twelve malarial paroxysms.

Results—

At this writing, fourteen patients have completed the course of treatments. Eleven have been relieved of intractable lightning pains and gastric crisis. These results were obtained after other active therapeutic measures had failed.

Other resistant tabetic symptoms such as cord bladders, paraesthesia and head pains were improved in the majority of the fourteen cases. Four of the patients were on the so-called "burnt out" list.

The author believes that this method of treating tabes dorsalis is the best method yet devised.

ROBERT HOROWITZ, '39

MULTIPLE ARTHRITIS IN PRESUMABLY TUBERCULOUS SUBJECTS: DIFFICULTIES IN DIAGNOSIS AND TREATMENT

By COLLINS, D. H. AND CAMERON, C.

Brit. J. Surg.; xxiv; 94; Oct., 1936

Diagnostic Inaccuracies—

Tuberculosis of a single large joint, e.g. hip or knee, in young persons before the age of puberty, can usually be diagnosed correctly after clinical and radiological examination. After puberty, other types of "non-specific" infective arthritis may make their appearance. In one series of 208 cases admitted for tuberculosis of the hip to the New York Orthopaedic Hospital, 22% were found to be wrongly diagnosed. In another series of 142 cases of proven tuberculosis, 38% were not so diagnosed.

Diagnostic Methods—

1. Guinea-pig inoculation of aspirated material: In a reported series of 24 tuberculous cases, animal inoculation failed to detect 3.

2. Histological Examination of Biopsy Specimens: In a reported series of 158

cases examination was correct in all but 3.2%.

3. X-Ray Diagnosis: X-ray is not of much value until a comparatively late stage when there is evidence of bone destruction.

(a) Decrease in the width of the joint space is a fairly early sign.

(b) In tuberculous arthritis the articular cartilage is often preserved at the points of contact of articul surfaces.

(c) If the joint space is maintained, in spite of associated bone destruction, there is a strong presumption of tuberculosis

(d) Tuberculin Reaction: A positive reaction is only presumptive of tuberculosis until all other possible active foci have been excluded. A negative reaction, in the absence of overwhelming tuberculous infection, advanced sepsis, anemia or other grave disease, can eliminate the diagnosis of tuberculosis with some certainty. Occasionally a marked focal reaction will occur in a tuberculous joint following a tuberculin test.

Early diagnosis is important because early immobilization might prevent destruction of bone in a tuberculous joint but in a non-specific arthritis would subject the patient to a long and expensive in-patient treatment and might result in an otherwise preventable ankylosis. Factors causing confusion in the differential diagnosis between Multiple Arthritis and Tuberculosis:

1. The insidious monarticular onset of some cases of multiple non-specific arthritis.

2. The co-existence in the patient of some visceral tuberculous lesion, which may or may not influence the course of a non-tuberculous polyarthritis.

3. The possible occurrence of a single tuberculous joint super-imposed upon a non-tuberculous polyarthritis.

4. The occasional incidence of true tuberculous arthritis in two or more joints.

5. The comparative infrequency of non-specific arthritis of the hip in patients under middle age, and the tendency to suppose such a condition to be tuberculous.

6. Modification of the course of non-specific arthritis due to early immobilization

7. The possibility that there exists an atypical tuberculous form of polyarthritis—tuberculous rheumatism.

In summarizing the unfavourable limitation of movement obtained following immobilization of suspected tuberculous joints, which later prove to be arthritic, the authors conclude that expectant treatment. consisting of general hygiene and rest in bed without immobilization should be adopted in all cases of joint disease until such time as they are definitely proved tuberculous.

F. M. COLE, '37

THE TREATMENT OF TORTICOLLIS

Clinic of Dr. Emil D. W. Houser Northwestern Medical School

Surg. Clin. N. Amer.; Feb., 1936

Torticollis or wryneck is due to a contracture of the muscles of the neck, chiefly the sternocleidomastoid, in which the head is drawn to the affected side and the chin is pointing to the opposite side.

There are two large groups of torticollis — congenital and acquired. The congenital type may be due to a muscular contracture or to a change in the cervical vertebrae. Of the acquired group, subdivisions may be made as follows:

(a) Scar contractures due to burns or lupus;
(b) Fascia contractures due to an inflammation;
(c) Muscle contractures due either to habit or injury; and
(d) Neurogenic, found chiefly in adults and spasmodic in nature.

Myogenic torticollis is the most common and occurs most often as the result of difficult labor. The right side is more frequently involved.

The first symptom is swelling which usually involves the lower one-third of the muscle. It is very tender and painful on motion. Later there is a definite contracture and fibrous formation. Over a long period of time the sheath and fascia become involved, asymmetry of the face and a scoliosis develop. The cause of this train of symptoms is believed to be a restriction of the circulation, such as occurs in Volkmann's contracture. There is often some hereditary factor.

Prognosis depends upon early recognition of the condition, prompt and adequate treatment and in which case results are excellent. If left untreated, the condition progresses until the face and skull become markedly asymmetrical and there is a scoliosis.

Treatment resolves itself into prophylactic and curative. The prophylactic treatment is essentially one of good obstetrical management. Curative treatment emphasizes earliest possible care.

Swelling and tenderness can be detected immediately after birth and if the condition is treated as an acute inflammatory process, excellent results are obtained. The part should be kept at rest in an over-corrected position—as being propped with pillows. Heat in the form of boracic compresses may be applied for 30 min., q. 8h. Massage is excellent and passive exercises should be carried out until there is no tendency toward the retraction. A Schanz bandage may be used if tne patient is near six months of age.

If the deformity is developed and the child is older, the open method of division of the sternocieidomastoid muscle is to be preferred. The skin is prepared and a small horizontal incision is made over the sternal and clavicular attachments. The platysma is divided; the attachment is resected. The head is manipulated to over-correction. The wound is closed in layers. A cast is applied which extends from the forehead, well down over the clavicles, and the head is maintained in an over-corrected position. The cast is removed in two to six weeks, depending upon the severity and the age of the patient.

After the cast is removed a Schanz bandage is used with forced extension and fixation of the head. Corrective exercises are essential. These are at first passive and then active. Unless exercises are carried out, recurrence is likely.

In cases of severe contracture in advanced cases, resection of the sternocleidomastoid muscle close to the mastoid process, may be done. After treatment is the same.

It is important to start the treatment as early as possible and to carry it out until every tendency for recurrence is obliterated.

B. LOYNES, '37

TREATMENT OF QUIESCENT TUMOUR ALBUS AND PSEUDARTHROSIS OF TUBERCULOUS ORIGIN IN CHILDREN

By DELAHAYE, A.

J. Bone and Jt. Surg., xviii; 1; 51-53

For the past seven years, at the Maritime Hospital in Berck, efforts have been directed toward the improvement of the technique and end-results in cases of tumour albus of the knee. When untreated or treated on conventional lines, these patients are left at the age at which resection is appropriate with very real disability, which has a tendency to increase.

In the estimation of the author, the following procedures have been found unsatisfactory in treatment of such cases:

1. Manipulation or osteotomy—merely palliative.
2. Intra-epiphyseal resection — proven unreliable by Broca.
3. Enucleation and filling of the epiphysis as done by Vignard.
4. The transepiphyseal grafts advocated by Robertson-Lavelle.

Two other procedures have given better results but necessitate opening the joint through tissues in which the disease is by no means extinct:

1. Intra-articular arthrodesis by transepiphyseal bone-pegging.
2. Juxta-epipnyseal arthrodesis by means of peripheral grafts — Frenell.

The new method advanced by Dr. Delahaye, and represented by a series of thirty cases, consists of a completely extra-articular method—anterior femoropatello-tibial arthrodesis. A long, supple, curved graft, of ample thickness is cut from the tibia of the healthy limb and is fixed at each end into two notches in the femur and tibia respectively, while, at the centre, it is slipped beneath two flaps cut from the anterior surface of the patella.

At the present time this operation is performed on only the chronic cases and in children in whom epiphyseal growth and ossification are relatively advanced.

A preliminary operation is carried out in those cases of severe flexional deformity of the knee—a posterior capsulotomy by anterior approach. By this means it is possible to divide the contracted posterior portion of the joint capsule at the level of the articular condyles and to obtain complete extension of the joint, without interference to the bones themselves.

Delahaye believes these various operative measures now provide adequate treatment for forms of the disease which hitherto have been treated only with imperfect success.

RUSSELL SCHRAM, 37

Editorial

THERE are still people today who would propose that the modern curriculum in medicine is not full enough. To such a suggestion, the suffering student would point out the enormity of his present task. He must, in six years, commit to memory most of the medical facts that are accepted today, and he will have heard mentioned, at least, all of them. In the study of anatomy, alone, the student must master fifteen thousand new words. To finally obtain an M. D. he must pass one hundred written examinations, thirty oral examinations, and innumerable quizzes, tutorials, and practical tests. Then, after struggling through one more examination—the Councils—he is ready for the practise of medicine.

Yet, we insist, his training is woefully incomplete. It is true, the successful candidate will be able to recognize and diagnose correctly that one-third of the patients that come to him that show typical forms of disease. He will even be able to alleviate the symptoms of that smaller fraction of these, for which a specific cure has been found. But what of the others? Is he trained to treat the vague symptom complexes, the functional cases, the "neurotics" and border-line psychotics who will make up two-thirds of his practise? The answer is easily found—in the obvious prosperity of chiropractors, osteopaths, in the palatial hotels at Williamsburg, in the electrical gadgets, with which reputable physicians desperately fill their offices. Does he obtain any instruction in those many other forms of non-medical advice that a doctor is called on daily to give his patients? Is any attempt made to give him that kindly, confidence inspiring mien which the laity refers to as "the bedside manner"? Is he encouraged to develop that competence in current affairs, in the cultural side of life, in the world of business, sport, music, art and literature, that will allow him to converse with ease of something besides "shop," that will put him at home with every class of people, and that will train him to take the prominent place in the community that his profession warrants?

These, of course, are things that cannot be learned in didactic lecture courses, that can't be plugged, as we plug so many other things (and forget them again, so easily). No—and we do not pretend to know the solution to the problem. But there are a few means which can be utilized to obtain that type of personality which will deal successfully with the difficulties of practise.

First—while we pursue the facts, the diseases, which are, of course,

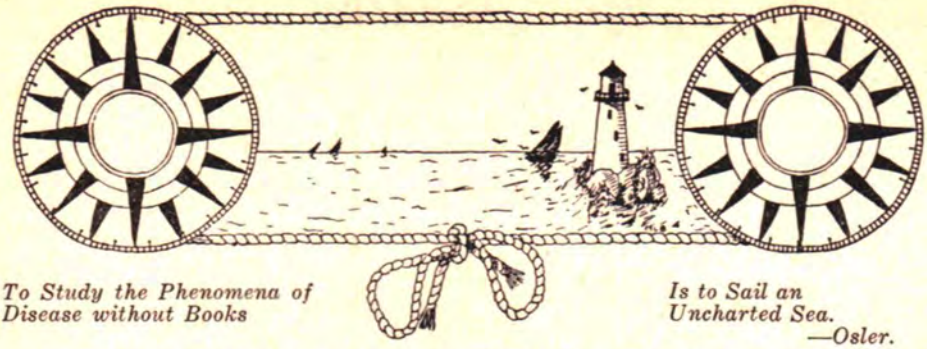
important, let us not forget that patients are people and not diseases. Any means which can train us to deal with people should be seized at. Activities like student government, societies, and sports, provide invaluable opportunities to cultivate the fine art of influencing people. Most precious of all the weapons of a doctor is the ability to speak confidently, and with ease; every student should make it a point of duty to learn to think on his feet while he is an undergraduate.

And, having acquired the self-confidence that knowledge brings, and the ability to express one's self without confusion—place one more course on your curriculum—put a copy of Osler's Aequanimitas by your bed, and read a bit every night.

THE GREAT DOCTOR

"Did you know Dr. Osler?" someone asked another. "Yes," was the answer, "intimately, but I only saw him once. It was late twilight; the city square was almost deserted when a woman carrying a heavy child came slowly up the square and sat down to rest on the coping bordering the pavement. The child's heavy head was pressed against her bosom and she seemed all in. I started to speak to her, when up the square came jauntily a man in full evening dress, top coat, silk hat, flower in his button-hole, light gloves in one hand and his cane in the other, evidently singing. In an instant, he saw the woman and her burden. He stopped, made a playful dive with his cane at the child. then throwing canes and gloves on the grass, he gently lifted the child into his arms, holding its head against his own breast as he talked to the mother. Then whistling to a little boy who chanced in sight, he said: 'Get a cab as quick as you can and if you are back in five minutes, riches! for you!' and he patted his breast pocket. The boy flew off and was back quickly with the cab. Dr. Osler put the woman in the cab, carefully placed the child on her lap—then he wrote on a card, 'This is Mrs. Osler's youngest. See that he is well taken care of until I come tomorrow night.' He read what he had written aloud to the woman, winked his eye at me, gave the driver his fare, told him to drive at once to the Hopkins Hospital, see that the woman and boy were safely attended to—then pressed a five-dollar bill in the woman's hand, said: 'Your laddie will be well looked after at the hospital. I will see him tomorrow. You go to your home and get drunk,' slammed the door of the cab and was off. All done while I was trying to say, 'Can I help you?' "

—*From* "THE GREAT PHYSICIAN," *by Edith G. Reid.*



To Study the Phenomena of Disease without Books

Is to Sail an Uncharted Sea.
—Osler.

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BACK FILES:—

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Biochemische Zeitschrift—To begin with Bd. 288.
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Surgery. A new publication—To begin with January, 1937.
Zentralblatt für Gynäkologie—To begin with Bd. 60, 1936.